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                   Patent Office Classifications
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 NEWS  9  SEP 01   INPADOC: New family current-awareness alert (SDI) available
 NEWS 10  SEP 01   New pricing for the Save Answers for SciFinder Wizard within
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 NEWS 11  SEP 01   New display format, HITSTR, available in WPIDS/WPINDEX/WPIX
 NEWS 12  SEP 14   STN Patent Forum to be held October 13, 2004, in Iselin, NJ
 NEWS 13  SEP 27   STANDARDS will no longer be available on STN
 NEWS 14  SEP 27   SWETSCAN will no longer be available on STN

 NEWS EXPRESS  JULY 30 CURRENT WINDOWS VERSION IS V7.01, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
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FILE 'HOME' ENTERED AT 14:39:55 ON 30 SEP 2004

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                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21
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FILE 'MEDLINE' ENTERED AT 14:40:25 ON 30 SEP 2004

FILE 'USPATFULL' ENTERED AT 14:40:25 ON 30 SEP 2004
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=> s haptoglobin-1 adj marker
   5 FILES SEARCHED...
L1   0 HAPTOGLOBIN-1 ADJ MARKER

=> s haptoglobin-1 precursor
   6 FILES SEARCHED...
L2   5 HAPTOGLOBIN-1 PRECURSOR

=> d l2 ti abs ibib tot

L2   ANSWER 1 OF 5   MEDLINE on STN
TI   Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AB   Screening for specific biomarkers of early-stage detection of ovarian cancer is a major health priority due to the asymptomatic nature and poor survival characteristic of the disease. We utilised two-dimensional gel electrophoresis (2DE) to identify differentially expressed proteins in the serum of ovarian cancer patients that may be useful as biomarkers of this disease. In this study, 38 ovarian cancer patients at different pathological grades (grade 1 (n=6), grade 2 (n=8) and grade 3 (n=24)) were compared to a control group of eight healthy women. Serum samples were treated with a mixture of Affigel-Blue and protein A (5 : 1) for 1 h to remove high abundance protein (e.g. immunoglobulin and albumin) and were displayed using 11 cm, pH 4-7 isoelectric focusing strips for the first dimension and 10% acrylamide gel electrophoresis for the second dimension. Protein spots were visualised by SYPRO-Ruby staining, imaged by FX-imager and compared and analysed by PDQuest software. A total of 24 serum proteins were differentially expressed in grade 1 (P<0.05), 31 in grade 2 (P<0.05) and 25 in grade 3 (P<0.05) ovarian cancer patients. Six of the protein spots that were significantly upregulated in all groups of ovarian cancer patients were identified by nano-electrospray quadrupole quadrupole time-of-flight mass spectrometry (n-ESIQ(q)TOFMS) and matrix-assisted laser desorption ionisation time-of-flight mass spectrometry (MALDI-TOFMS) as isoforms of **haptoglobin-1 precursor** (HAP1), a liver glycoprotein present in human serum. Further identification of the spots at different pathological grades was confirmed by Western blotting using monoclonal antibody against a haptoglobin epitope contained within HAP1. Immunohistochemical localisation of HAP1-like activity was present in malignant ovarian epithelium and stroma but strong immunostaining was present in blood vessels, areas with myxomatous stroma and vascular spaces. No tissue localisation of

HAP1-like immunoreactivity was observed in normal ovarian surface epithelium. These data highlight the need to assess circulating concentration of HAP1 in the serum of ovarian cancer patients and evaluate its potential as a biomarker in the early diagnosis of ovarian cancer.

ACCESSION NUMBER: 2004323790 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15199385
TITLE: Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AUTHOR: Ahmed N; Barker G; Oliva K T; Hoffmann P; Riley C; Reeve S; Smith A I; Kemp B E; Quinn M A; Rice G E
CORPORATE SOURCE: Gynaecological Cancer Research Centre, Royal Women's Hospital, 132 Grattan Street, Carlton, Victoria 3053, Australia.. nuzhata@unimelb.edu.au
SOURCE: British journal of cancer, (2004 Jul 5) 91 (1) 129-40. Journal code: 0370635. ISSN: 0007-0920.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200408
ENTRY DATE: Entered STN: 20040701
Last Updated on STN: 20040807
Entered Medline: 20040806

L2 ANSWER 2 OF 5 USPATFULL on STN
TI Non-genetic based protein disease markers
AB Protein disease markers for obesity, osteoporosis, diabetes, osteoarthritis and hypertension are disclosed. These markers are not inherited or of genetic origin as they were not found in identical twins of the affected individual. Methods and uses for diagnostic, therapeutic and drug discovery are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:141506 USPATFULL
TITLE: Non-genetic based protein disease markers
INVENTOR(S): Myers, Timothy G., Kensington, MD, UNITED STATES
Pieper, Rembert, Washington, DC, UNITED STATES
Taylor, John, JR., Clayton, NC, UNITED STATES
Steiner, Sandra, Gaithersburg, MD, UNITED STATES
Anderson, N. Leigh, Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002072492	A1	20020613
APPLICATION INFO.:	US 2001-886271	A1	20010622 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-660242, filed on 12 Sep 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Dean H. Nakamura, Roylance Abrams Berdo & Goodman, 1300 19th Street, N.W., Washington, DC, 20036
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 1425
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 3 OF 5 USPATFULL on STN
TI Nucleic acid molecules encoding human protease homologs
AB The invention relates to polynucleotides encoding newly identified protease homologs. The invention also relates to the proteases. The invention further relates to methods using the protease polypeptides and polynucleotides as a target for diagnosis and treatment in

protease-mediated disorders. The invention further relates to drug-screening methods using the protease polypeptides and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the protease polypeptides and polynucleotides. The invention further relates to procedures for producing the protease polypeptides and polynucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2002:122764 USPATFULL		
TITLE:	Nucleic acid molecules encoding human protease homologs		
INVENTOR(S):	Robison, Keith E., Wilmington, MA, United States		
PATENT ASSIGNEE(S):	Millennium Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6395889	B1	20020528
APPLICATION INFO.:	US 1999-392184		19990909 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Achutamurthy, Ponnathapu		
ASSISTANT EXAMINER:	Moore, William W.		
LEGAL REPRESENTATIVE:	Alston & Bird LLP		
NUMBER OF CLAIMS:	1		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	0 Drawing Figure(s); 0 Drawing Page(s)		
LINE COUNT:	5266		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 4 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.

AB Screening for specific biomarkers of early-stage detection of ovarian cancer is a major health priority due to the asymptomatic nature and poor survival characteristic of the disease. We utilised two-dimensional gel electrophoresis (2DE) to identify differentially expressed proteins in the serum of ovarian cancer patients that may be useful as biomarkers of this disease. In this study, 38 ovarian cancer patients at different pathological grades (grade 1 (n = 6), grade 2 (n = 8) and grade 3 (n = 24)) were compared to a control group of eight healthy women. Serum samples were treated with a mixture of Affigel-Blue and protein A (5:1) for 1 h to remove high abundance protein (e.g. immunoglobulin and albumin) and were displayed using 11 cm, pH 4-7 isoelectric focusing strips for the first dimension and 10% acrylamide gel electrophoresis for the second dimension. Protein spots were visualised by SYPRO-Ruby staining, imaged by FX-imager and compared and analysed by PDQuest software. A total of 24 serum proteins were differentially expressed in grade 1 ($P < 0.05$), 31 in grade 2 ($P < 0.05$) and 25 in grade 3 ($P < 0.05$) ovarian cancer patients. Six of the protein spots that were significantly upregulated in all groups of ovarian cancer patients were identified by nano-electrospray quadrupole quadrupole time-of-flight mass spectrometry (n-ESIQ(q)TOFMS) and matrix-assisted laser desorption ionisation time-of-flight mass spectrometry (MALDI-TOFMS) as isoforms of **haptoglobin-1 precursor** (HAP1), a liver glycoprotein present in human serum. Further identification of the spots at different pathological grades was confirmed by Western blotting using monoclonal antibody against a haptoglobin epitope contained within HAP1. Immunohistochemical localisation of HAP1-like activity was present in malignant ovarian epithelium and stroma but strong immunostaining was present in blood vessels, areas with myxomatous stroma and vascular spaces. No tissue localisation of HAP1-like immunoreactivity was observed in normal ovarian surface epithelium. These data highlight the need to assess circulating

concentration of HAP1 in the serum of ovarian cancer patients and evaluate its potential as a biomarker in the early diagnosis of ovarian cancer. .COPYRGT. 2004 Cancer Research UK.

ACCESSION NUMBER: 2004331760 EMBASE
TITLE: Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AUTHOR: Ahmed N.; Barker G.; Oliva K.T.; Hoffmann P.; Riley C.; Reeve S.; Smith Al.; Kemp B.E.; Quinn M.A.; Rice G.E.
CORPORATE SOURCE: Dr. N. Ahmed, Gynaecological Cancer Res. Centre, Royal Women's Hospital, 132 Grattan Street, Carlton, Vic. 3053, Australia. nuzhata@unimelb.edu.au
SOURCE: British Journal of Cancer, (5 Jul 2004) 91/1 (129-140). Refs: 32
ISSN: 0007-0920 CODEN: BJCAAI
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
010 Obstetrics and Gynecology
016 Cancer
027 Biophysics, Bioengineering and Medical Instrumentation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L2 ANSWER 5 OF 5 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AB Screening for specific biomarkers of early-stage detection of ovarian cancer is a major health priority due to the asymptomatic nature and poor survival characteristic of the disease. We utilised two-dimensional gel electrophoresis (2DE) to identify differentially expressed proteins in the serum of ovarian cancer patients that may be useful as biomarkers of this disease. In this study, 38 ovarian cancer patients at different pathological grades (grade 1 (n = 6), grade 2 (n = 8) and grade 3 (n = 24)) were compared to a control group of eight healthy women. Serum samples were treated with a mixture of Affigel-Blue and protein A (5 : 1) for 1 h to remove high abundance protein (e. g. immunoglobulin and albumin) and were displayed using 11 cm, pH 4-7 isoelectric focusing strips for the first dimension and 10% acrylamide gel electrophoresis for the second dimension. Protein spots were visualised by SYPRO-Ruby staining, imaged by FX-imager and compared and analysed by PDQuest software. A total of 24 serum proteins were differentially expressed in grade 1 (P 0.05), 31 in grade 2 (P 0.05) and 25 in grade 3 (P 0.05) ovarian cancer patients. Six of the protein spots that were significantly upregulated in all groups of ovarian cancer patients were identified by nano-electrospray quadrupole quadrupole time-of-flight mass spectrometry (n-ESIQ(q)TOFMS) and matrix-assisted laser desorption ionisation time-of-flight mass spectrometry (MALDI-TOFMS) as isoforms of **haptoglobin-1 precursor** (HAP1), a liver glycoprotein present in human serum. Further identification of the spots at different pathological grades was confirmed by Western blotting using monoclonal antibody against a haptoglobin epitope contained within HAP1. Immunohistochemical localisation of HAP1-like activity was present in malignant ovarian epithelium and stroma but strong immunostaining was present in blood vessels, areas with myxomatous stroma and vascular spaces. No tissue localisation of HAP1-like immunoreactivity was observed in normal ovarian surface epithelium. These data highlight the need to assess circulating concentration of HAP1 in the serum of ovarian cancer patients and evaluate its potential as a biomarker in the early diagnosis of ovarian cancer.
ACCESSION NUMBER: 2004:379522 BIOSIS
DOCUMENT NUMBER: PREV200400378224

TITLE: Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AUTHOR(S): Ahmed, N. [Reprint Author]; Barker, G.; Oliva, K. T.; Hoffmann, P.; Riley, C.; Reeve, S.; Smith, A. I.; Kemp, B. E.; Quinn, M. A.; Rice, G. E.
CORPORATE SOURCE: Gynaecol Canc Res Ctr, Royal Hosp Women, 132 Grattan St, Carlton, Vic, 3053, Australia
nuzhata@unimelb.edu.au
SOURCE: British Journal of Cancer, (July 5 2004) Vol. 91, No. 1, pp. 129-140. print.
ISSN: 0007-0920 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Sep 2004
Last Updated on STN: 22 Sep 2004

=> s immunoglobulin M heavy chain
L3 123 IMMUNOGLOBULIN M HEAVY CHAIN

=> s SNC73
L4 15 SNC73

=> s l3 and hypertension
L5 4 L3 AND HYPERTENSION

=> d l4 ti abs ibib tot

L4 ANSWER 1 OF 15 MEDLINE on STN
TI Expression and recombination mechanism of **SNC73** (IgHalpha1) in human epithelial cancer cell line.
AB OBJECTIVE: To study if the gene **SNC73** (IgHalpha1) is expressed in human epithelial cancer cell line and to interpret the recombination mechanism. METHODS: Human epithelial cancer cells of SW480 line were cultured. RT-PCR and Western blotting were used to examine the expression of **SNC73**, recombination activating gene 1 (RAG1), and RAG2. The RT-PCR products were confirmed by sequencing. Immunohistochemistry was used to detect the expression of IgHalpha1, Igkappa, and Iglambda in these epithelial cancer cells. RESULTS: The human epithelial cancer cell line (SW480) positively expressed **SNC73**, RAG1, and RAG2. IgHalpha1 and Igkappa was strongly expressed in SW480 cells, but Iglambda was undetectable. The sequence of the constant region of **SNC73** in SW480 cells is identical to that of IgA1. Both sequencing and Western blotting showed that the RAG1 and RAG2 expressed in SW480 cells were identical to that expressed in pre-B lymphocytes. CONCLUSION: Immunoglobulin alpha-1 gene is expressed in non-lymphoid cells, which may be a potential genetic marker for the development of colorectal cancer. Recombination signal sequence (RSS)-mediated recombination may take part in the rearrangement of immunoglobulin alpha-1 gene in human epithelial cancer cell line.
ACCESSION NUMBER: 2003461977 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 14521728
TITLE: Expression and recombination mechanism of **SNC73** (IgHalpha1) in human epithelial cancer cell line.
AUTHOR: Geng Li-Yi; Zheng Shu; Peng Jia-Ping
CORPORATE SOURCE: Cancer Institute, Zhejiang University, Hangzhou 310009, China.
SOURCE: Zhonghua yi xue za zhi, (2003 Sep 10) 83 (17) 1493-6.
Journal code: 7511141. ISSN: 0376-2491.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 20031003
Last Updated on STN: 20031218

L4 ANSWER 2 OF 15 MEDLINE on STN
TI Expression of a novel immunoglobulin gene **SNC73** in human cancer and non-cancerous tissues.
AB AIM: To investigate the expression of immunoglobulin gene **SNC73** in malignant tumors and non-cancerous normal tissues. METHODS: Expression level of **SNC73** in tumors and non-cancerous tissues from the same patient was determined by reverse transcription polymerase chain reaction and enzyme-linked immunosorbent assay (RT-PCR-ELISA) in 90 cases of malignant tumors, including colorectal cancer, gastric cancer, breast cancer, lung cancer and liver cancer. Analysis on the correlation of **SNC73** expression with sex, age, site, grade of differentiation, depth of invasion, and metastases in colorectal cancer patients was made. RESULTS: Expression level of **SNC73** in non-cancerous colorectal mucosa and colorectal cancerous tissues was 1.234+/-0.842 and 0.737+/-0.731, respectively ($P<0.01$), with the mean ratio of 7.134+/-14.092 (range, 0.36-59.54). Expression of **SNC73** showed no significant difference among gastric cancer, breast cancer, lung cancer and liver cancer when compared with non-cancerous tissues ($P>0.05$). No correlation was found between **SNC73** expression level and various clinicopathological factors, including sex, age, site, grade of differentiation, depth of invasion and metastases of CRC patients. CONCLUSION: Down-regulation of **SNC73** expression may be a relatively specific phenomenon in colorectal cancer. **SNC73** is a potential genetic marker for the carcinongenesis of colorectal cancer. The relationship of **SNC73** expression and carcinogenesis of colorectal cancer merits further study.
ACCESSION NUMBER: 2003198056 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12717855
TITLE: Expression of a novel immunoglobulin gene **SNC73** in human cancer and non-cancerous tissues.
AUTHOR: Hu Jian-Bin; Zheng Shu; Deng Yong-Chuan
CORPORATE SOURCE: Department of Radiation Oncology, Sir Run Run Shaw Hospital, Zhejiang University Medical College, Hangzhou, Zhejiang Province, China.
SOURCE: World journal of gastroenterology : WJG, (2003 May) 9 (5) 1054-7.
Journal code: 100883448. ISSN: 1007-9327.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 20030429
Last Updated on STN: 20030715
Entered Medline: 20030714

L4 ANSWER 3 OF 15 MEDLINE on STN
TI Expression of a novel immunoglobulin gene **SNC73** in human cancer and its significance.
AB OBJECTIVE: To investigate the expression of a new immunoglobulin gene **SNC73** in malignant tumor and normal tissue and its significance. METHODS: Expression level of **SNC73** in tumors and normal tissues was determined by reverse transcription-polymerase chain reaction and enzyme-linked immunosorbent assay (RT-PCR-ELISA) in 90 malignant tumors, including colorectal cancer, gastric cancer, breast cancer, lung cancer and liver cancer. Analysis on relation of **SNC73** expression with age, sex, site, differentiation grade, depth of invasion and metastasis of colorectal cancer was made to assess the clinical significance of **SNC73**. RESULTS: Mean ratio of **SNC73** expression level in normal mucosa and colorectal cancer tissue was 7.134 ($P < 0.01$). Expression of **SNC73** showed no significant difference among

gastric cancer, breast cancer, lung cancer or liver cancer as compared with the control normal tissues (P > 0.05). CONCLUSION: Down-regulation of **SNC73** expression is a relatively specific phenomenon in colorectal cancer for which development **SNC73** may be a potential genetic marker. The study on relationship of **SNC73** expression with development of colorectal cancer is promising.

ACCESSION NUMBER: 2002240080 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11977634
TITLE: Expression of a novel immunoglobulin gene **SNC73** in human cancer and its significance.
AUTHOR: Hu Jianbin; Deng Yongchuan; Zheng Shu
CORPORATE SOURCE: Cancer Institute, Zhejiang University, Hangzhou 310009, China.
SOURCE: Zhonghua zhong liu za zhi [Chinese journal of oncology], (2002 Jan) 24 (1) 38-40.
Journal code: 7910681. ISSN: 0253-3766.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 20020430
Last Updated on STN: 20020516
Entered Medline: 20020515

L4 ANSWER 4 OF 15 MEDLINE on STN
TI Structure and expression of colorectal cancer related Immunoglobulin novel gene **SNC73**.
AB OBJECTIVE: To study the structure and function of a colorectal cancer-associated gene **SNC73** obtained by subtractive hybridization technique. METHODS: Direct sequencing was performed on cDNA of **SNC73** gene. In situ-max fluorescence in situ hybridization was used in chromosome mapping of **SNC73**. Expression of **SNC73** in various cancer cell lines and differential expression between normal mucosa and colorectal cancer tissue were examined by Northern blotting and RT-PCR. Expression of **SNC73** in colorectal epithelium was detected by in situ hybridization and in situ PCR. RESULTS: Open reading frame prediction showed that **SNC73** encodes a peptide identical to the constant region of an IgA molecule in the carboxyl-terminus. The gene was mapped to human chromosome 14q32. The expression of **SNC73** in colorectal cancer tissue and that in normal mucosa was different (P < 0.05). **SNC73** was lowly expressed in colorectal epithelium. CONCLUSION: Decrease in **SNC73** expression may be a potential genetic marker for the development of colorectal cancer. An immunoglobulin alpha-1 gene can be expressed in non-lymphoid cells.

ACCESSION NUMBER: 2002073324 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11798924
TITLE: Structure and expression of colorectal cancer related Immunoglobulin novel gene **SNC73**.
AUTHOR: Zheng S; Cao J; Geng L
CORPORATE SOURCE: Cancer Institute, Zhejiang University, Hangzhou 310009, China.
SOURCE: Zhonghua yi xue za zhi, (2001 Apr 25) 81 (8) 485-8.
Journal code: 7511141. ISSN: 0376-2491.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF067420
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020430
Entered Medline: 20020429

L4 ANSWER 5 OF 15 USPATFULL on STN

TI Proteins and nucleic acids encoding same

AB Disclosed herein are nucleic acid sequences that encode novel polypeptides. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivatives, variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2004:44501 USPATFULL

TITLE: Proteins and nucleic acids encoding same

INVENTOR(S): Tchernev, Velizar T., Branford, CT, UNITED STATES
Spytek, Kimberly A., New Haven, CT, UNITED STATES
Zerhusen, Bryan D., Branford, CT, UNITED STATES
Patturajan, Meera, Branford, CT, UNITED STATES
Shimkets, Richard A., West Haven, CT, UNITED STATES
Li, Li, Branford, CT, UNITED STATES
Gangolli, Esha A., Madison, CT, UNITED STATES
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Anderson, David W., Branford, CT, UNITED STATES
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Taupier, Raymond J., JR., East Haven, CT, UNITED STATES
Gusev, Vladimir Y., UNITED STATES
Colman, Steven D., Guilford, CT, UNITED STATES
Wolenc, Adam Ryan, New Haven, CT, UNITED STATES
Pena, Carol E. A., Guilford, CT, UNITED STATES
Furtak, Katarzyna, Anosia, CT, UNITED STATES
Grosse, William M., Bransford, CT, UNITED STATES
Alsobrook, John P., II, Madison, CT, UNITED STATES
Lepley, Denise M., Branford, CT, UNITED STATES
Rieger, Daniel K., Branford, CT, UNITED STATES
Burgess, Catherine E., Wethersfield, CT, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004033493	A1	20040219	
APPLICATION INFO.:	US 2002-72012	A1	20020131	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-267459P	20010208	(60)
	US 2001-266975P	20010207	(60)
	US 2001-267057P	20010207	(60)
	US 2001-266767P	20010205	(60)
	US 2001-266406P	20010202	(60)
	US 2001-265395P	20010131	(60)
	US 2001-265412P	20010131	(60)
	US 2001-265517P	20010131	(60)
	US 2001-265514P	20010131	(60)
	US 2001-267823P	20010209	(60)
	US 2001-268974P	20010215	(60)
	US 2001-271855P	20010227	(60)
	US 2001-271839P	20010227	(60)
	US 2001-273046P	20010302	(60)
	US 2001-272788P	20010302	(60)
	US 2001-275989P	20010314	(60)
	US 2001-275925P	20010314	(60)
	US 2001-275947P	20010314	(60)

US 2001-275950P	20010314	(60)
US 2001-276450P	20010315	(60)
US 2001-276448P	20010315	(60)
US 2001-276397P	20010316	(60)
US 2001-276768P	20010316	(60)
US 2001-278652P	20010320	(60)
US 2001-278775P	20010326	(60)
US 2001-278778P	20010326	(60)
US 2001-279882P	20010329	(60)
US 2001-279884P	20010329	(60)
US 2001-280147P	20010330	(60)
US 2001-283083P	20010411	(60)
US 2001-282992P	20010411	(60)
US 2001-285133P	20010420	(60)
US 2001-285749P	20010423	(60)
US 2001-288327P	20010503	(60)
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US 2001-294047P	20010529	(60)
US 2001-294473P	20010530	(60)
US 2001-296964P	20010608	(60)
US 2001-298959P	20010618	(60)
US 2001-299324P	20010619	(60)
US 2001-312020P	20010813	(60)
US 2001-312908P	20010816	(60)
US 2001-312889P	20010816	(60)
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US 2001-315470P	20010828	(60)
US 2001-316447P	20010831	(60)
US 2001-318115P	20010907	(60)
US 2001-318118P	20010907	(60)
US 2001-318740P	20010912	(60)
US 2001-323379P	20010919	(60)
US 2001-330308P	20011018	(60)
US 2001-330245P	20011018	(60)
US 2001-332701P	20011114	(60)
US 2001-271664P	20010226	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Ivor R. Elrifi, Ph.D., Mintz, Levin, Cohn, Ferris,, Glovsky and Popeo, P.C., One Financial Center, Boston, MA, 02111
NUMBER OF CLAIMS: 49
EXEMPLARY CLAIM: 1
LINE COUNT: 59681
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 6 OF 15 USPATFULL on STN
TI Classification and prognosis prediction of acute lymphoblastic leukemia by gene expression profiling
AB The present invention provides methods and compositions useful for diagnosing and choosing treatment for leukemia patients. The claimed methods include methods of assigning a subject affected by leukemia to a leukemia risk group, methods of predicting whether a subject affected by leukemia has an increased risk of relapse, methods of predicting whether a subject affected by leukemia has an increased risk of developing secondary acute myeloid leukemia, methods to aid in the determination of a prognosis for a subject affected by leukemia, methods of choosing a therapy for a subject affected by leukemia, and methods of monitoring the disease state in a subject undergoing one or more therapies for leukemia. The claimed compositions include arrays having capture probes for the differentially-expressed genes of the invention, computer readable media having digitally-encoded expression profiles associated with leukemia risk groups, and kits for diagnosing and choosing therapy for leukemia patients.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:24674 USPATFULL
TITLE:	Classification and prognosis prediction of acute lymphoblastic leukemia by gene expression profiling
INVENTOR(S):	Downing, James R., Cordova, TN, UNITED STATES Yeoh, Eng-Juh, Singapore, SINGAPORE Wilkins, Dawn E., Oxford, MS, UNITED STATES Wong, Limsoon, Singapore, SINGAPORE

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004018513	A1	20040129	
APPLICATION INFO.:	US 2003-391271	A1	20030318	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-367144P	20020322 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	ALSTON AND BIRD LLP, ST. JUDE CHILDREN'S RESEARCH HOSPITAL, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS:	64
EXEMPLARY CLAIM:	1
LINE COUNT:	9169

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 7 OF 15 USPATFULL on STN

TI Method for the detection of gene transcripts in blood and uses thereof

AB The present invention is directed to detection and measurement of gene transcripts in blood. Specifically provided is a RT-PCR analysis performed on a drop of blood for detecting, diagnosing and monitoring diseases using tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-associated genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:18757 USPATFULL
TITLE:	Method for the detection of gene transcripts in blood and uses thereof
INVENTOR(S):	Liew, Choong-Chin, Toronto, CANADA

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004014059	A1	20040122	
APPLICATION INFO.:	US 2002-268730	A1	20021009	(10)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2000-477148, filed on 4 Jan 2000, ABANDONED			

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-115125P	19990106 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	Randolph Ted Apple, Morrison & Foerster LLP, 755 Page Mill Road, Palo Alto, CA, 94304-1018
NUMBER OF CLAIMS:	24
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	7 Drawing Page(s)
LINE COUNT:	5099

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 8 OF 15 USPATFULL on STN

TI Methods of diagnosis of ovarian cancer, compositions and methods of screening for modulators of ovarian cancer

AB Described herein are genes whose expression are up-regulated or down-regulated in ovarian cancer. Related methods and compositions that can be used for diagnosis and treatment of ovarian cancer are disclosed. Also described herein are methods that can be used to identify modulators of ovarian cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:7329 USPATFULL			
TITLE:	Methods of diagnosis of ovarian cancer, compositions and methods of screening for modulators of ovarian cancer			
INVENTOR(S):	Mack, David H., Menlo Park, CA, UNITED STATES Gish, Kurt C., San Francisco, CA, UNITED STATES			
PATENT ASSIGNEE(S):	Eos Biotechnology, Inc., South San Francisco, CA (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004005563	A1	20040108	
APPLICATION INFO.:	US 2002-173999	A1	20020617	(10)
	NUMBER		DATE	
PRIORITY INFORMATION:	US 2002-372246P		20020412	(60)
	US 2001-350666P		20011113	(60)
	US 2001-315287P		20010827	(60)
	US 2001-299234P		20010618	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834			
NUMBER OF CLAIMS:	24			
EXEMPLARY CLAIM:	1			
LINE COUNT:	32540			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 9 OF 15 USPATFULL on STN

TI Novel methods of diagnosis of metastatic colorectal cancer, compositions and methods of screening for modulators of metastatic colorectal cancer

AB Described herein are methods and compositions that can be used for diagnosis and treatment of metastatic colorectal cancer. Also described herein are methods that can be used to identify modulators of metastatic colorectal cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:334944 USPATFULL			
TITLE:	Novel methods of diagnosis of metastatic colorectal cancer, compositions and methods of screening for modulators of metastatic colorectal cancer			
INVENTOR(S):	Mack, David H., Menlo Park, CA, UNITED STATES Markowitz, Sanford David, Pepper Pike, OH, UNITED STATES			
PATENT ASSIGNEE(S):	Eos Biotechnology, Inc., South San Francisco, CA (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003235820	A1	20031225	
APPLICATION INFO.:	US 2002-87080	A1	20020227	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-284555P	20010417 (60)
	US 2001-281149P	20010402 (60)
	US 2001-272206P	20010227 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
LINE COUNT: 22670
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 10 OF 15 USPATFULL on STN
TI Genes expressed in colon cancer
AB The present invention relates to a combination comprising a plurality of cDNAs which are differentially expressed in colon cancer and which may be used in their entirety or in part as to diagnose, to stage to treat or to monitor the progression or treatment of colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:106194 USPATFULL
TITLE: Genes expressed in colon cancer
INVENTOR(S): Lasek, Amy K.W., Oakland, CA, UNITED STATES
Sornasse, Thierry, Mountain View, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003073105	A1	20030417	
APPLICATION INFO.:	US 2002-158646	A1	20020529	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-295239P	20010531 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LEGAL DEPARTMENT, INCYTE GENOMICS, INC., 3160 PORTER DRIVE, PALO ALTO, CA, 94304
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 4837
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 11 OF 15 USPATFULL on STN
TI Non-genetic based protein disease markers
AB Protein disease markers for obesity, osteoporosis, diabetes, osteoarthritis and hypertension are disclosed. These markers are not inherited or of genetic origin as they were not found in identical twins of the affected individual. Methods and uses for diagnostic, therapeutic and drug discovery are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:141506 USPATFULL
TITLE: Non-genetic based protein disease markers
INVENTOR(S): Myers, Timothy G., Kensington, MD, UNITED STATES
Pieper, Rembert, Washington, DC, UNITED STATES
Taylor, John, JR., Clayton, NC, UNITED STATES
Steiner, Sandra, Gaithersburg, MD, UNITED STATES
Anderson, N. Leigh, Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002072492	A1	20020613

APPLICATION INFO.: US 2001-886271 A1 20010622 (9)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-660242, filed on 12 Sep 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Dean H. Nakamura, Roylance Abrams Berdo & Goodman, 1300 19th Street, N.W., Washington, DC, 20036
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 1425
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 12 OF 15 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Novel combination of cDNAs which are differentially expressed in colon cancer, useful for detecting differential expression of one or more cDNAs in a sample containing nucleic acid samples.
AN AAD59167 cDNA DGENE
AB The present invention relates to combination of cDNAs which are differentially expressed in colon cancer. The invention is useful for producing and purifying antibody, utilized as markers for treatment efficacy against colon cancer. The invention is also useful for gene therapy. The present sequence is human **SNC73** protein (**SNC73**) cDNA
ACCESSION NUMBER: AAD59167 cDNA DGENE
TITLE: Novel combination of cDNAs which are differentially expressed in colon cancer, useful for detecting differential expression of one or more cDNAs in a sample containing nucleic acid samples.
INVENTOR: Lasek A K W; Sornasse T
PATENT ASSIGNEE: (LASE-I) LASEK A K W.
(SORN-I) SORNASSE T.
PATENT INFO: US 2003073105 A1 20030417 88p
APPLICATION INFO: US 2002-158646 20020529
PRIORITY INFO: US 2001-295239P 20010531
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2003-605964 [57]
DESCRIPTION: Human **SNC73** protein (**SNC73**) cDNA.

L4 ANSWER 13 OF 15 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Diagnosing and monitoring prostate disorders, by analysis of 26 gene transcripts that exhibit aberrant expression levels in prostate disorder tissues, and provides a means of early diagnosis -
AN AAD07360 DNA DGENE
AB The patent discloses a method for diagnosing, prognosing or monitoring a prostate disorder which involves the analysis of 26 gene transcripts (referred as markers) that exhibit aberrant expression levels in prostate disorder tissues and provides a means of early diagnosis. This method is useful for diagnosing, prognosing or monitoring a prostate disorder. It also provides a means of distinguishing prostate cancer from benign prostatic hyperplasia (BPH) and for identifying potential anti-prostate disorder therapeutic compounds. The present sequence is a human DNA encoding **SNC73** protein (referred as marker 11). The **SNC73** sequence is identified as an mRNA downregulated in colorectal cancer.
ACCESSION NUMBER: AAD07360 DNA DGENE
TITLE: Diagnosing and monitoring prostate disorders, by analysis of 26 gene transcripts that exhibit aberrant expression levels in prostate disorder tissues, and provides a means of early diagnosis -
INVENTOR: Bull J H; Ellison G; Paskins L D
PATENT ASSIGNEE: (ASTR) ASTRAZENECA AB.
(ASTR) ASTRAZENECA UK LTD.

PATENT INFO: WO 2001036674 A2 20010525 69p
APPLICATION INFO: WO 2000-GB4267 20001108
PRIORITY INFO: GB 1999-26805 19991113
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-343837 [36]
DESCRIPTION: Human DNA encoding **SNC73** protein (marker 11).

L4 ANSWER 14 OF 15 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Expression of a novel immunoglobulin gene **SNC73** in human cancer and non-cancerous tissues.
AB Aim: To investigate the expression of immunoglobulin gene **SNC73** in malignant tumors and non-cancerous normal tissues. Methods: Expression level of **SNC73** in tumors and non- cancerous tissues from the same patient was determined by reverse transcription polymerase chain reaction and enzyme-linked immunosorbent assay (RT-PCR-ELISA) in 90 cases of malignant tumors, including colorectal cancer, gastric cancer, breast cancer, lung cancer and liver cancer. Analysis on the correlation of **SNC73** expression with sex, age, site, grade of differentiation, depth of invasion, and metastases in colorectal cancer patients was made. Results: Expression level of **SNC73** in non-cancerous colorectal mucosa and colorectal cancerous tissues was 1.234±0.842 and 0.737±0.731, respectively ($P<0.01$), with the mean ratio of 7.134±14.092 (range, 0.36-59.54). Expression of **SNC73** showed no significant difference among gastric cancer, breast cancer, lung cancer and liver cancer when compared with non-cancerous tissues ($P>0.05$). No correlation was found between **SNC73** expression level and various clinicopathological factors, including sex, age, site, grade of differentiation, depth of invasion and metastases of CRC patients. Conclusion: Down-regulation of **SNC73** expression may be a relatively specific phenomenon in colorectal cancer. **SNC73** is a potential genetic marker for the carcinogenesis of colorectal cancer. The relationship of **SNC73** expression and carcinogenesis of colorectal cancer merits further study.
ACCESSION NUMBER: 2003228655 EMBASE
TITLE: Expression of a novel immunoglobulin gene **SNC73** in human cancer and non-cancerous tissues.
AUTHOR: Hu J.-B.; Zheng S.; Deng Y.-C.
CORPORATE SOURCE: S. Zheng, Cancer Institute, Zhejiang University, Hangzhou 310009, Zhejiang Province, China. zhengshu@mail.hz.zj.cn
SOURCE: World Journal of Gastroenterology, (15 May 2003) 9/5 (1054-1057).
Refs: 34
ISSN: 1007-9327 CODEN: WJGAF2
COUNTRY: China
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 022 Human Genetics
016 Cancer
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

L4 ANSWER 15 OF 15 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Expression of a novel immunoglobulin gene **SNC73** in human cancer and its significance.
AB Objective To investigate the expression of a new immunoglobulin gene **SNC73** in malignant tumor and normal tissue and its significance. Methods Expression level of **SNC73** in tumors and normal tissues was determined by reverse transcription-polymerase chain reaction and enzyme-linked immunosorbent assay (RT-PCR-ELISA) in 90 malignant tumors, including colorectal cancer, gastric cancer, breast cancer, lung cancer and liver cancer. Analysis on relation of **SNC73** expression with

age, sex, site, differentiation grade, depth of invasion and metastasis of colorectal cancer was made to assess the clinical significance of **SNC73**. Results Mean ratio of **SNC73** expression level in normal mucosa and colorectal cancer tissue was 7.134 ($P < 0.01$). Expression of **SNC73** showed no significant difference among gastric cancer, breast cancer, lung cancer or liver cancer as compared with the control normal tissues ($P > 0.05$). Conclusion Down-regulation of **SNC73** expression is a relatively specific phenomenon in colorectal cancer for which development **SNC73** may be a potential genetic marker. The study on relationship of **SNC73** expression with development of colorectal cancer is promising.

ACCESSION NUMBER: 2002:296037 BIOSIS
DOCUMENT NUMBER: PREV200200296037
TITLE: Expression of a novel immunoglobulin gene **SNC73** in human cancer and its significance.
AUTHOR(S): Hu Jianbin [Reprint author]; Deng Yongchuan [Reprint author]; Zheng Shu [Reprint author]
CORPORATE SOURCE: Cancer Institute, Zhejiang University, Hangzhou, 310009, China
SOURCE: Zhonghua Zhongliu Zazhi, (January, 2002) Vol. 24, No. 1, pp. 38-40. print.
CODEN: CCLCDY. ISSN: 0253-3766.
DOCUMENT TYPE: Article
LANGUAGE: Chinese
ENTRY DATE: Entered STN: 15 May 2002
Last Updated on STN: 15 May 2002

=> d l5 ti abs ibib tot

L5 ANSWER 1 OF 4 USPATFULL on STN
TI Gene sequence variations with utility in determining the treatment of disease, in genes relating to drug processing
AB Methods for identifying and utilizing variances in genes relating to efficacy and safety of medical therapy and other aspects of medical therapy are described, including methods for selecting an effective treatment.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:221287 USPATFULL
TITLE: Gene sequence variations with utility in determining the treatment of disease, in genes relating to drug processing
INVENTOR(S): Stanton, Vincent P., JR., Belmont, MA, UNITED STATES
PATENT ASSIGNEE(S): Variagenics, Inc., a Delaware corporation (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004171056	A1	20040902
APPLICATION INFO.:	US 2004-798873	A1	20040311 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-648123, filed on 25 Aug 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-590783, filed on 8 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-501955, filed on 10 Feb 2000, ABANDONED Continuation-in-part of Ser. No. WO 2000-US1392, filed on 20 Jan 2000, PENDING Continuation-in-part of Ser. No. US 1999-451252, filed on 29 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-427835, filed on 26 Oct 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-414330, filed on 6 Oct 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-389993, filed on 3 Sep 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-370841, filed

on 9 Aug 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-300747, filed on 26 Apr 1999, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-121047P	19990222 (60)
	US 1999-131334P	19990426 (60)
	US 1999-131191P	19990426 (60)
	US 1999-139440P	19990615 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
LINE COUNT: 11893
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 2 OF 4 USPATFULL on STN
TI Tumor necrosis factor receptor 2
AB The present disclosure describes the use of genetic variance information for genes involved in inflammatory or immunologic disease, disorder, or dysfunction. The variance information is indicative of the expected response of a patient to a method of treatment. Methods of determining relevant variance information and additional methods of using such variance information are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:4504 USPATFULL
TITLE: Tumor necrosis factor receptor 2
INVENTOR(S): Stanton, Jr., Vincent P., Belmont, MA, United States
PATENT ASSIGNEE(S): Nuvelo, Inc., Sunnyvale, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6673908	B1	20040106
APPLICATION INFO.:	US 2001-968455		20011001 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-649035, filed on 25 Aug 2000 Continuation-in-part of Ser. No. US 2000-590749, filed on 8 Jun 2000, now abandoned Continuation-in-part of Ser. No. US 2000-495780, filed on 1 Feb 2000, now abandoned Continuation-in-part of Ser. No. US 2000-492712, filed on 27 Jan 2000, now abandoned Continuation-in-part of Ser. No. WO 2000-US1392, filed on 20 Jan 2000 Continuation-in-part of Ser. No. US 968455 Continuation-in-part of Ser. No. US 1999-451252, filed on 29 Nov 1999, now abandoned Continuation-in-part of Ser. No. US 1999-427835, filed on 26 Oct 1999, now abandoned Continuation-in-part of Ser. No. US 1999-414330, filed on 6 Oct 1999, now abandoned Continuation-in-part of Ser. No. US 1999-389993, filed on 3 Sep 1999, now abandoned Continuation-in-part of Ser. No. US 1999-370841, filed on 9 Aug 1999, now abandoned Continuation-in-part of Ser. No. US 1999-300747, filed on 26 Apr 1999, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-131334P	19990426 (60)
	US 1999-131191P	19990426 (60)
	US 1999-121047P	19990222 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Benzion, Gary
ASSISTANT EXAMINER: Chakrabarti, Arun Kr.
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 17463
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 3 OF 4 USPATFULL on STN
TI Non-genetic based protein disease markers
AB Protein disease markers for obesity, osteoporosis, diabetes, osteoarthritis and **hypertension** are disclosed. These markers are not inherited or of genetic origin as they were not found in identical twins of the affected individual. Methods and uses for diagnostic, therapeutic and drug discovery are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:141506 USPATFULL
TITLE: Non-genetic based protein disease markers
INVENTOR(S): Myers, Timothy G., Kensington, MD, UNITED STATES
Pieper, Rembert, Washington, DC, UNITED STATES
Taylor, John, JR., Clayton, NC, UNITED STATES
Steiner, Sandra, Gaithersburg, MD, UNITED STATES
Anderson, N. Leigh, Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002072492	A1	20020613
APPLICATION INFO.:	US 2001-886271	A1	20010622 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-660242, filed on 12 Sep 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Dean H. Nakamura, Roylance Abrams Berdo & Goodman, 1300 19th Street, N.W., Washington, DC, 20036
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 1425
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 4 OF 4 USPATFULL on STN
TI Nucleic acids, proteins and antibodies
AB This invention relates to newly identified tissue specific cancer associated polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cancer antigens," and to the complete gene sequences associated therewith and to the expression products thereof, as well as the use of such tissue specific cancer antigens for detection, prevention and treatment of tissue specific disorders, particularly the presense of cancer. This invention relates to the cancer antigens as well as vectors, host cells, antibodies directed to cancer antigens and recombinant and synthetic methods for producing the same. Also provided are diagnostic methods for diagnosing and treating, preventing and/or prognosing tissue specific disorders, including cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of cancer antigens of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and/or function of the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:99407 USPATFULL
TITLE: Nucleic acids, proteins and antibodies
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002052308	A1	20020502
APPLICATION INFO.:	US 2001-925301	A1	20010810 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. WO 2000-US5882, filed on 8 Mar 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-124270P	19990312 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
LINE COUNT: 30577
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> s HAP-1 and hypertension
6 FILES SEARCHED...
L6 7 HAP-1 AND HYPERTENSION

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L6 ANSWER 1 OF 7 USPATFULL on STN
TI IL-1 gene cluster and associated inflammatory polymorphisms and haplotypes
AB The invention provides methods and compositions relating to identification and use of genetic information from the IL-1 gene cluster--including the structure and organization of novel IL-1-like genes found within the IL-1 locus as well as polymorphisms and associated haplotypes within these genes. The invention thereby expands the repertoire of useful genetic information available from the IL-1 locus--which contains the previously-identified IL-1α, IL-1β and IL-1RN genes, for predicting IL-1 associated phenotypes (e.g. increased or decreased risks of inflammatory disease) and for treating IL-1 haplotype associated inflammatory phenotypes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:221269 USPATFULL
TITLE: IL-1 gene cluster and associated inflammatory polymorphisms and haplotypes
INVENTOR(S): Nicklin, Martin, Sheffield, UNITED KINGDOM
Duff, Gordon, Sheffield, UNITED KINGDOM
Kornman, Kenneth, Newton, MA, UNITED STATES
Kolpin, Maryam Rafie, Medford, MA, UNITED STATES
Hsieh, Chung-Ming, West Roxbury, MA, UNITED STATES
Govindaraju, Raju, Lexington, MA, UNITED STATES
Aziz, Nazneen, Lexington, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004171038	A1	20040902
APPLICATION INFO.:	US 2003-716029	A1	20031117 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2003-351702, filed on 27 Jan 2003, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-351951P	20020125 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Ivor R. Elrifi, Mintz, Levin, Cohn, Ferris., Glovsky and Popeo, P.C., One Financial Center, Boston, MA, 02111
NUMBER OF CLAIMS: 39
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 37 Drawing Page(s)
LINE COUNT: 3495
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 2 OF 7 USPATFULL on STN
TI Schizophrenia associated genes, proteins and biallelic markers
AB The invention concerns the human sbg1, g34665, sbg2, g35017 and g35018 genes, polynucleotides, polypeptides biallelic markers, and human chromosome 13q31-q33 biallelic markers. The invention also concerns the association established between schizophrenia and bipolar disorder and the biallelic markers and the sbg1, g34665, sbg2, g35017 and g35018 genes and nucleotide sequences. The invention provides means to identify compounds useful in the treatment of schizophrenia, bipolar disorder and related diseases, means to determine the predisposition of individuals to said disease as well as means for the disease diagnosis and prognosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:312153 USPATFULL
TITLE: Schizophrenia associated genes, proteins and biallelic markers
INVENTOR(S): Cohen, Daniel, Paris, FRANCE
Blumenfeld, Marta, Paris, FRANCE
Chumakov, Ilya, Vaux-le-Penil, FRANCE
Bougueleret, Lydie, Petit Lancy, SWITZERLAND
Bihain, Bernard, Cancale, FRANCE
Essioux, Laurent, Paris, FRANCE
PATENT ASSIGNEE(S): GENSET, S.A., Paris, FRANCE (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003219750	A1	20031127
APPLICATION INFO.:	US 2002-147603	A1	20020516 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-539333, filed on 30 Mar 2000, GRANTED, Pat. No. US 6476208 Continuation-in-part of Ser. No. US 1999-416384, filed on 12 Oct 1999, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-126903P	19990330 (60)
	US 1999-131971P	19990430 (60)
	US 1999-132065P	19990430 (60)
	US 1999-143928P	19990714 (60)
	US 1999-145915P	19990727 (60)
	US 1999-146453P	19990729 (60)
	US 1999-146452P	19990729 (60)
	US 1999-162288P	19991028 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SALIWANCHIK LLOYD & SALIWANCHIK, A PROFESSIONAL ASSOCIATION, 2421 N.W. 41ST STREET, SUITE A-1, GAINESVILLE, FL, 326066669
NUMBER OF CLAIMS: 50

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=> s haptoglobin-1 adj marker
   5 FILES SEARCHED...
L1   0 HAPTOGLOBIN-1 ADJ MARKER

=> s haptoglobin-1 precursor
   6 FILES SEARCHED...
L2   5 HAPTOGLOBIN-1 PRECURSOR

=> d l2 ti abs ibib tot

L2   ANSWER 1 OF 5   MEDLINE on STN
TI   Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AB   Screening for specific biomarkers of early-stage detection of ovarian cancer is a major health priority due to the asymptomatic nature and poor survival characteristic of the disease. We utilised two-dimensional gel electrophoresis (2DE) to identify differentially expressed proteins in the serum of ovarian cancer patients that may be useful as biomarkers of this disease. In this study, 38 ovarian cancer patients at different pathological grades (grade 1 (n=6), grade 2 (n=8) and grade 3 (n=24)) were compared to a control group of eight healthy women. Serum samples were treated with a mixture of Affigel-Blue and protein A (5 : 1) for 1 h to remove high abundance protein (e.g. immunoglobulin and albumin) and were displayed using 11 cm, pH 4-7 isoelectric focusing strips for the first dimension and 10% acrylamide gel electrophoresis for the second dimension. Protein spots were visualised by SYPRO-Ruby staining, imaged by FX-imager and compared and analysed by PDQuest software. A total of 24 serum proteins were differentially expressed in grade 1 (P<0.05), 31 in grade 2 (P<0.05) and 25 in grade 3 (P<0.05) ovarian cancer patients. Six of the protein spots that were significantly upregulated in all groups of ovarian cancer patients were identified by nano-electrospray quadrupole quadrupole time-of-flight mass spectrometry (n-ESIQ(q)TOFMS) and matrix-assisted laser desorption ionisation time-of-flight mass spectrometry (MALDI-TOFMS) as isoforms of **haptoglobin-1 precursor** (HAP1), a liver glycoprotein present in human serum. Further identification of the spots at different pathological grades was confirmed by Western blotting using monoclonal antibody against a haptoglobin epitope contained within HAP1. Immunohistochemical localisation of HAP1-like activity was present in malignant ovarian epithelium and stroma but strong immunostaining was present in blood vessels, areas with myxomatous stroma and vascular spaces. No tissue localisation of

HAP1-like immunoreactivity was observed in normal ovarian surface epithelium. These data highlight the need to assess circulating concentration of HAP1 in the serum of ovarian cancer patients and evaluate its potential as a biomarker in the early diagnosis of ovarian cancer.

ACCESSION NUMBER: 2004323790 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15199385
TITLE: Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AUTHOR: Ahmed N; Barker G; Oliva K T; Hoffmann P; Riley C; Reeve S; Smith A I; Kemp B E; Quinn M A; Rice G E
CORPORATE SOURCE: Gynaecological Cancer Research Centre, Royal Women's Hospital, 132 Grattan Street, Carlton, Victoria 3053, Australia.. nuzhata@unimelb.edu.au
SOURCE: British journal of cancer, (2004 Jul 5) 91 (1) 129-40. Journal code: 0370635. ISSN: 0007-0920.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200408
ENTRY DATE: Entered STN: 20040701
Last Updated on STN: 20040807
Entered Medline: 20040806

L2 ANSWER 2 OF 5 USPATFULL on STN
TI Non-genetic based protein disease markers
AB Protein disease markers for obesity, osteoporosis, diabetes, osteoarthritis and hypertension are disclosed. These markers are not inherited or of genetic origin as they were not found in identical twins of the affected individual. Methods and uses for diagnostic, therapeutic and drug discovery are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:141506 USPATFULL
TITLE: Non-genetic based protein disease markers
INVENTOR(S): Myers, Timothy G., Kensington, MD, UNITED STATES
Pieper, Rembert, Washington, DC, UNITED STATES
Taylor, John, JR., Clayton, NC, UNITED STATES
Steiner, Sandra, Gaithersburg, MD, UNITED STATES
Anderson, N. Leigh, Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002072492	A1	20020613
APPLICATION INFO.:	US 2001-886271	A1	20010622 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-660242, filed on 12 Sep 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Dean H. Nakamura, Roylance Abrams Berdo & Goodman, 1300 19th Street, N.W., Washington, DC, 20036
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 1425
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 3 OF 5 USPATFULL on STN
TI Nucleic acid molecules encoding human protease homologs
AB The invention relates to polynucleotides encoding newly identified protease homologs. The invention also relates to the proteases. The invention further relates to methods using the protease polypeptides and polynucleotides as a target for diagnosis and treatment in

protease-mediated disorders. The invention further relates to drug-screening methods using the protease polypeptides and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the protease polypeptides and polynucleotides. The invention further relates to procedures for producing the protease polypeptides and polynucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2002:122764 USPATFULL		
TITLE:	Nucleic acid molecules encoding human protease homologs		
INVENTOR(S):	Robison, Keith E., Wilmington, MA, United States		
PATENT ASSIGNEE(S):	Millennium Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6395889	B1	20020528
APPLICATION INFO.:	US 1999-392184		19990909 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Achutamurthy, Ponnathapu		
ASSISTANT EXAMINER:	Moore, William W.		
LEGAL REPRESENTATIVE:	Alston & Bird LLP		
NUMBER OF CLAIMS:	1		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	0 Drawing Figure(s); 0 Drawing Page(s)		
LINE COUNT:	5266		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 4 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.

AB Screening for specific biomarkers of early-stage detection of ovarian cancer is a major health priority due to the asymptomatic nature and poor survival characteristic of the disease. We utilised two-dimensional gel electrophoresis (2DE) to identify differentially expressed proteins in the serum of ovarian cancer patients that may be useful as biomarkers of this disease. In this study, 38 ovarian cancer patients at different pathological grades (grade 1 (n = 6), grade 2 (n = 8) and grade 3 (n = 24)) were compared to a control group of eight healthy women. Serum samples were treated with a mixture of Affigel-Blue and protein A (5:1) for 1 h to remove high abundance protein (e.g. immunoglobulin and albumin) and were displayed using 11 cm, pH 4-7 isoelectric focusing strips for the first dimension and 10% acrylamide gel electrophoresis for the second dimension. Protein spots were visualised by SYPRO-Ruby staining, imaged by FX-imager and compared and analysed by PDQuest software. A total of 24 serum proteins were differentially expressed in grade 1 ($P < 0.05$), 31 in grade 2 ($P < 0.05$) and 25 in grade 3 ($P < 0.05$) ovarian cancer patients. Six of the protein spots that were significantly upregulated in all groups of ovarian cancer patients were identified by nano-electrospray quadrupole quadrupole time-of-flight mass spectrometry (n-ESIQ(q)TOFMS) and matrix-assisted laser desorption ionisation time-of-flight mass spectrometry (MALDI-TOFMS) as isoforms of **haptoglobin-1 precursor** (HAP1), a liver glycoprotein present in human serum. Further identification of the spots at different pathological grades was confirmed by Western blotting using monoclonal antibody against a haptoglobin epitope contained within HAP1. Immunohistochemical localisation of HAP1-like activity was present in malignant ovarian epithelium and stroma but strong immunostaining was present in blood vessels, areas with myxomatous stroma and vascular spaces. No tissue localisation of HAP1-like immunoreactivity was observed in normal ovarian surface epithelium. These data highlight the need to assess circulating

concentration of HAP1 in the serum of ovarian cancer patients and evaluate its potential as a biomarker in the early diagnosis of ovarian cancer.

ACCESSION NUMBER: 2004331760 EMBASE
TITLE: Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AUTHOR: Ahmed N.; Barker G.; Oliva K.T.; Hoffmann P.; Riley C.; Reeve S.; Smith Al.; Kemp B.E.; Quinn M.A.; Rice G.E.
CORPORATE SOURCE: Dr. N. Ahmed, Gynaecological Cancer Res. Centre, Royal Women's Hospital, 132 Grattan Street, Carlton, Vic. 3053, Australia. nuzhata@unimelb.edu.au
SOURCE: British Journal of Cancer, (5 Jul 2004) 91/1 (129-140). Refs: 32
ISSN: 0007-0920 CODEN: BJCAAI
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
010 Obstetrics and Gynecology
016 Cancer
027 Biophysics, Bioengineering and Medical Instrumentation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L2 ANSWER 5 OF 5 BIOSIS
TI Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AB Screening for specific biomarkers of early-stage detection of ovarian cancer is a major health priority due to the asymptomatic nature and poor survival characteristic of the disease. We utilised two-dimensional gel electrophoresis (2DE) to identify differentially expressed proteins in the serum of ovarian cancer patients that may be useful as biomarkers of this disease. In this study, 38 ovarian cancer patients at different pathological grades (grade 1 (n = 6), grade 2 (n = 8) and grade 3 (n = 24)) were compared to a control group of eight healthy women. Serum samples were treated with a mixture of Affigel-Blue and protein A (5 : 1) for 1 h to remove high abundance protein (e. g. immunoglobulin and albumin) and were displayed using 11 cm, pH 4-7 isoelectric focusing strips for the first dimension and 10% acrylamide gel electrophoresis for the second dimension. Protein spots were visualised by SYPRO-Ruby staining, imaged by FX-imager and compared and analysed by PDQuest software. A total of 24 serum proteins were differentially expressed in grade 1 (P 0.05), 31 in grade 2 (P 0.05) and 25 in grade 3 (P 0.05) ovarian cancer patients. Six of the protein spots that were significantly upregulated in all groups of ovarian cancer patients were identified by nano-electrospray quadrupole quadrupole time-of-flight mass spectrometry (n-ESIQ(q)TOFMS) and matrix-assisted laser desorption ionisation time-of-flight mass spectrometry (MALDI-TOFMS) as isoforms of **haptoglobin-1 precursor** (HAP1), a liver glycoprotein present in human serum. Further identification of the spots at different pathological grades was confirmed by Western blotting using monoclonal antibody against a haptoglobin epitope contained within HAP1. Immunohistochemical localisation of HAP1-like activity was present in malignant ovarian epithelium and stroma but strong immunostaining was present in blood vessels, areas with myxomatous stroma and vascular spaces. No tissue localisation of HAP1-like immunoreactivity was observed in normal ovarian surface epithelium. These data highlight the need to assess circulating concentration of HAP1 in the serum of ovarian cancer patients and evaluate its potential as a biomarker in the early diagnosis of ovarian cancer.
ACCESSION NUMBER: 2004:379522 BIOSIS
DOCUMENT NUMBER: PREV200400378224

TITLE: Proteomic-based identification of **haptoglobin-1 precursor** as a novel circulating biomarker of ovarian cancer.
AUTHOR(S): Ahmed, N. [Reprint Author]; Barker, G.; Oliva, K. T.; Hoffmann, P.; Riley, C.; Reeve, S.; Smith, A. I.; Kemp, B. E.; Quinn, M. A.; Rice, G. E.
CORPORATE SOURCE: Gynaecol Canc Res Ctr, Royal Hosp Women, 132 Grattan St, Carlton, Vic, 3053, Australia
nuzhata@unimelb.edu.au
SOURCE: British Journal of Cancer, (July 5 2004) Vol. 91, No. 1, pp. 129-140. print.
ISSN: 0007-0920 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Sep 2004
Last Updated on STN: 22 Sep 2004

=> s immunoglobulin M heavy chain
L3 123 IMMUNOGLOBULIN M HEAVY CHAIN

=> s SNC73
L4 15 SNC73

=> s l3 and hypertension
L5 4 L3 AND HYPERTENSION

=> d l4 ti abs ibib tot

L4 ANSWER 1 OF 15 MEDLINE on STN
TI Expression and recombination mechanism of **SNC73** (IgHalpha1) in human epithelial cancer cell line.
AB OBJECTIVE: To study if the gene **SNC73** (IgHalpha1) is expressed in human epithelial cancer cell line and to interpret the recombination mechanism. METHODS: Human epithelial cancer cells of SW480 line were cultured. RT-PCR and Western blotting were used to examine the expression of **SNC73**, recombination activating gene 1 (RAG1), and RAG2. The RT-PCR products were confirmed by sequencing. Immunohistochemistry was used to detect the expression of IgHalpha1, Igkappa, and Iglambda in these epithelial cancer cells. RESULTS: The human epithelial cancer cell line (SW480) positively expressed **SNC73**, RAG1, and RAG2. IgHalpha1 and Igkappa was strongly expressed in SW480 cells, but Iglambda was undetectable. The sequence of the constant region of **SNC73** in SW480 cells is identical to that of IgA1. Both sequencing and Western blotting showed that the RAG1 and RAG2 expressed in SW480 cells were identical to that expressed in pre-B lymphocytes. CONCLUSION: Immunoglobulin alpha-1 gene is expressed in non-lymphoid cells, which may be a potential genetic marker for the development of colorectal cancer. Recombination signal sequence (RSS)-mediated recombination may take part in the rearrangement of immunoglobulin alpha-1 gene in human epithelial cancer cell line.
ACCESSION NUMBER: 2003461977 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 14521728
TITLE: Expression and recombination mechanism of **SNC73** (IgHalpha1) in human epithelial cancer cell line.
AUTHOR: Geng Li-Yi; Zheng Shu; Peng Jia-Ping
CORPORATE SOURCE: Cancer Institute, Zhejiang University, Hangzhou 310009, China.
SOURCE: Zhonghua yi xue za zhi, (2003 Sep 10) 83 (17) 1493-6.
Journal code: 7511141. ISSN: 0376-2491.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 20031003
Last Updated on STN: 20031218

L4 ANSWER 2 OF 15 MEDLINE on STN
TI Expression of a novel immunoglobulin gene **SNC73** in human cancer and non-cancerous tissues.
AB AIM: To investigate the expression of immunoglobulin gene **SNC73** in malignant tumors and non-cancerous normal tissues. METHODS: Expression level of **SNC73** in tumors and non-cancerous tissues from the same patient was determined by reverse transcription polymerase chain reaction and enzyme-linked immunosorbent assay (RT-PCR-ELISA) in 90 cases of malignant tumors, including colorectal cancer, gastric cancer, breast cancer, lung cancer and liver cancer. Analysis on the correlation of **SNC73** expression with sex, age, site, grade of differentiation, depth of invasion, and metastases in colorectal cancer patients was made. RESULTS: Expression level of **SNC73** in non-cancerous colorectal mucosa and colorectal cancerous tissues was 1.234+/-0.842 and 0.737+/-0.731, respectively (P<0.01), with the mean ratio of 7.134+/-14.092 (range, 0.36-59.54). Expression of **SNC73** showed no significant difference among gastric cancer, breast cancer, lung cancer and liver cancer when compared with non-cancerous tissues (P>0.05). No correlation was found between **SNC73** expression level and various clinicopathological factors, including sex, age, site, grade of differentiation, depth of invasion and metastases of CRC patients. CONCLUSION: Down-regulation of **SNC73** expression may be a relatively specific phenomenon in colorectal cancer. **SNC73** is a potential genetic marker for the carcinongenesis of colorectal cancer. The relationship of **SNC73** expression and carcinogenesis of colorectal cancer merits further study.
ACCESSION NUMBER: 2003198056 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12717855
TITLE: Expression of a novel immunoglobulin gene **SNC73** in human cancer and non-cancerous tissues.
AUTHOR: Hu Jian-Bin; Zheng Shu; Deng Yong-Chuan
CORPORATE SOURCE: Department of Radiation Oncology, Sir Run Run Shaw Hospital, Zhejiang University Medical College, Hangzhou, Zhejiang Province, China.
SOURCE: World journal of gastroenterology : WJG, (2003 May) 9 (5) 1054-7.
Journal code: 100883448. ISSN: 1007-9327.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 20030429
Last Updated on STN: 20030715
Entered Medline: 20030714

L4 ANSWER 3 OF 15 MEDLINE on STN
TI Expression of a novel immunoglobulin gene **SNC73** in human cancer and its significance.
AB OBJECTIVE: To investigate the expression of a new immunoglobulin gene **SNC73** in malignant tumor and normal tissue and its significance. METHODS: Expression level of **SNC73** in tumors and normal tissues was determined by reverse transcription-polymerase chain reaction and enzyme-linked immunosorbent assay (RT-PCR-ELISA) in 90 malignant tumors, including colorectal cancer, gastric cancer, breast cancer, lung cancer and liver cancer. Analysis on relation of **SNC73** expression with age, sex, site, differentiation grade, depth of invasion and metastasis of colorectal cancer was made to assess the clinical significance of **SNC73**. RESULTS: Mean ratio of **SNC73** expression level in normal mucosa and colorectal cancer tissue was 7.134 (P < 0.01). Expression of **SNC73** showed no significant difference among

gastric cancer, breast cancer, lung cancer or liver cancer as compared with the control normal tissues (P > 0.05). CONCLUSION: Down-regulation of **SNC73** expression is a relatively specific phenomenon in colorectal cancer for which development **SNC73** may be a potential genetic marker. The study on relationship of **SNC73** expression with development of colorectal cancer is promising.

ACCESSION NUMBER: 2002240080 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11977634
TITLE: Expression of a novel immunoglobulin gene **SNC73** in human cancer and its significance.
AUTHOR: Hu Jianbin; Deng Yongchuan; Zheng Shu
CORPORATE SOURCE: Cancer Institute, Zhejiang University, Hangzhou 310009, China.
SOURCE: Zhonghua zhong liu za zhi [Chinese journal of oncology], (2002 Jan) 24 (1) 38-40.
Journal code: 7910681. ISSN: 0253-3766.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 20020430
Last Updated on STN: 20020516
Entered Medline: 20020515

L4 ANSWER 4 OF 15 MEDLINE on STN
TI Structure and expression of colorectal cancer related Immunoglobulin novel gene **SNC73**.
AB OBJECTIVE: To study the structure and function of a colorectal cancer-associated gene **SNC73** obtained by subtractive hybridization technique. METHODS: Direct sequencing was performed on cDNA of **SNC73** gene. In situ-max fluorescence in situ hybridization was used in chromosome mapping of **SNC73**. Expression of **SNC73** in various cancer cell lines and differential expression between normal mucosa and colorectal cancer tissue were examined by Northern blotting and RT-PCR. Expression of **SNC73** in colorectal epithelium was detected by in situ hybridization and in situ PCR. RESULTS: Open reading frame prediction showed that **SNC73** encodes a peptide identical to the constant region of an IgA molecule in the carboxyl-terminus. The gene was mapped to human chromosome 14q32. The expression of **SNC73** in colorectal cancer tissue and that in normal mucosa was different (P < 0.05). **SNC73** was lowly expressed in colorectal epithelium. CONCLUSION: Decrease in **SNC73** expression may be a potential genetic marker for the development of colorectal cancer. An immunoglobulin alpha-1 gene can be expressed in non-lymphoid cells.

ACCESSION NUMBER: 2002073324 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11798924
TITLE: Structure and expression of colorectal cancer related Immunoglobulin novel gene **SNC73**.
AUTHOR: Zheng S; Cao J; Geng L
CORPORATE SOURCE: Cancer Institute, Zhejiang University, Hangzhou 310009, China.
SOURCE: Zhonghua yi xue za zhi, (2001 Apr 25) 81 (8) 485-8.
Journal code: 7511141. ISSN: 0376-2491.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF067420
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020430
Entered Medline: 20020429

L4 ANSWER 5 OF 15 USPATFULL on STN
TI Proteins and nucleic acids encoding same
AB Disclosed herein are nucleic acid sequences that encode novel polypeptides. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivatives, variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2004:44501 USPATFULL
TITLE: Proteins and nucleic acids encoding same
INVENTOR(S): Tchernev, Velizar T., Branford, CT, UNITED STATES
Spytek, Kimberly A., New Haven, CT, UNITED STATES
Zerhusen, Bryan D., Branford, CT, UNITED STATES
Patturajan, Meera, Branford, CT, UNITED STATES
Shimkets, Richard A., West Haven, CT, UNITED STATES
Li, Li, Branford, CT, UNITED STATES
Gangolli, Esha A., Madison, CT, UNITED STATES
Padigaru, Muralidhara, Branford, CT, UNITED STATES
Anderson, David W., Branford, CT, UNITED STATES
Rastelli, Luca, Guilford, CT, UNITED STATES
Miller, Charles E., Hill Drive, CT, UNITED STATES
Gerlach, Valerie, Branford, CT, UNITED STATES
Taupier, Raymond J., JR., East Haven, CT, UNITED STATES
Gusev, Vladimir Y., UNITED STATES
Colman, Steven D., Guilford, CT, UNITED STATES
Wolenc, Adam Ryan, New Haven, CT, UNITED STATES
Pena, Carol E. A., Guilford, CT, UNITED STATES
Furtak, Katarzyna, Anosia, CT, UNITED STATES
Grosse, William M., Bransford, CT, UNITED STATES
Alsobrook, John P., II, Madison, CT, UNITED STATES
Lepley, Denise M., Branford, CT, UNITED STATES
Rieger, Daniel K., Branford, CT, UNITED STATES
Burgess, Catherine E., Wethersfield, CT, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004033493	A1	20040219	
APPLICATION INFO.:	US 2002-72012	A1	20020131	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-267459P	20010208 (60)
	US 2001-266975P	20010207 (60)
	US 2001-267057P	20010207 (60)
	US 2001-266767P	20010205 (60)
	US 2001-266406P	20010202 (60)
	US 2001-265395P	20010131 (60)
	US 2001-265412P	20010131 (60)
	US 2001-265517P	20010131 (60)
	US 2001-265514P	20010131 (60)
	US 2001-267823P	20010209 (60)
	US 2001-268974P	20010215 (60)
	US 2001-271855P	20010227 (60)
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	US 2001-273046P	20010302 (60)
	US 2001-272788P	20010302 (60)
	US 2001-275989P	20010314 (60)
	US 2001-275925P	20010314 (60)
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US 2001-276450P	20010315 (60)
US 2001-276448P	20010315 (60)
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US 2001-313930P	20010821 (60)
US 2001-315470P	20010828 (60)
US 2001-316447P	20010831 (60)
US 2001-318115P	20010907 (60)
US 2001-318118P	20010907 (60)
US 2001-318740P	20010912 (60)
US 2001-323379P	20010919 (60)
US 2001-330308P	20011018 (60)
US 2001-330245P	20011018 (60)
US 2001-332701P	20011114 (60)
US 2001-271664P	20010226 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Ivor R. Elrifi, Ph.D., Mintz, Levin, Cohn, Ferris,, Glovsky and Popeo, P.C., One Financial Center, Boston, MA, 02111
NUMBER OF CLAIMS: 49
EXEMPLARY CLAIM: 1
LINE COUNT: 59681
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 6 OF 15 USPATFULL on STN

TI Classification and prognosis prediction of acute lymphoblastic leukemia by gene expression profiling

AB The present invention provides methods and compositions useful for diagnosing and choosing treatment for leukemia patients. The claimed methods include methods of assigning a subject affected by leukemia to a leukemia risk group, methods of predicting whether a subject affected by leukemia has an increased risk of relapse, methods of predicting whether a subject affected by leukemia has an increased risk of developing secondary acute myeloid leukemia, methods to aid in the determination of a prognosis for a subject affected by leukemia, methods of choosing a therapy for a subject affected by leukemia, and methods of monitoring the disease state in a subject undergoing one or more therapies for leukemia. The claimed compositions include arrays having capture probes for the differentially-expressed genes of the invention, computer readable media having digitally-encoded expression profiles associated with leukemia risk groups, and kits for diagnosing and choosing therapy for leukemia patients.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:24674 USPATFULL
TITLE:	Classification and prognosis prediction of acute lymphoblastic leukemia by gene expression profiling
INVENTOR(S):	Downing, James R., Cordova, TN, UNITED STATES Yeoh, Eng-Juh, Singapore, SINGAPORE Wilkins, Dawn E., Oxford, MS, UNITED STATES Wong, Limsoon, Singapore, SINGAPORE

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004018513	A1	20040129
APPLICATION INFO.:	US 2003-391271	A1	20030318 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-367144P	20020322 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	ALSTON AND BIRD LLP, ST. JUDE CHILDREN'S RESEARCH HOSPITAL, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS:	64
EXEMPLARY CLAIM:	1
LINE COUNT:	9169

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 7 OF 15 USPATFULL on STN

TI Method for the detection of gene transcripts in blood and uses thereof

AB The present invention is directed to detection and measurement of gene transcripts in blood. Specifically provided is a RT-PCR analysis performed on a drop of blood for detecting, diagnosing and monitoring diseases using tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-associated genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:18757 USPATFULL
TITLE:	Method for the detection of gene transcripts in blood and uses thereof
INVENTOR(S):	Liew, Choong-Chin, Toronto, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004014059	A1	20040122
APPLICATION INFO.:	US 2002-268730	A1	20021009 (10)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2000-477148, filed on 4 Jan 2000, ABANDONED		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-115125P	19990106 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	Randolph Ted Apple, Morrison & Foerster LLP, 755 Page Mill Road, Palo Alto, CA, 94304-1018
NUMBER OF CLAIMS:	24
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	7 Drawing Page(s)
LINE COUNT:	5099

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 8 OF 15 USPATFULL on STN

TI Methods of diagnosis of ovarian cancer, compositions and methods of screening for modulators of ovarian cancer

AB Described herein are genes whose expression are up-regulated or down-regulated in ovarian cancer. Related methods and compositions that can be used for diagnosis and treatment of ovarian cancer are disclosed. Also described herein are methods that can be used to identify modulators of ovarian cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:7329 USPATFULL			
TITLE:	Methods of diagnosis of ovarian cancer, compositions and methods of screening for modulators of ovarian cancer			
INVENTOR(S):	Mack, David H., Menlo Park, CA, UNITED STATES Gish, Kurt C., San Francisco, CA, UNITED STATES			
PATENT ASSIGNEE(S):	Eos Biotechnology, Inc., South San Francisco, CA (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004005563	A1	20040108	
APPLICATION INFO.:	US 2002-173999	A1	20020617	(10)
	NUMBER		DATE	
PRIORITY INFORMATION:	US 2002-372246P		20020412	(60)
	US 2001-350666P		20011113	(60)
	US 2001-315287P		20010827	(60)
	US 2001-299234P		20010618	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834			
NUMBER OF CLAIMS:	24			
EXEMPLARY CLAIM:	1			
LINE COUNT:	32540			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 9 OF 15 USPATFULL on STN

TI Novel methods of diagnosis of metastatic colorectal cancer, compositions and methods of screening for modulators of metastatic colorectal cancer

AB Described herein are methods and compositions that can be used for diagnosis and treatment of metastatic colorectal cancer. Also described herein are methods that can be used to identify modulators of metastatic colorectal cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:334944 USPATFULL			
TITLE:	Novel methods of diagnosis of metastatic colorectal cancer, compositions and methods of screening for modulators of metastatic colorectal cancer			
INVENTOR(S):	Mack, David H., Menlo Park, CA, UNITED STATES Markowitz, Sanford David, Pepper Pike, OH, UNITED STATES			
PATENT ASSIGNEE(S):	Eos Biotechnology, Inc., South San Francisco, CA (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003235820	A1	20031225	
APPLICATION INFO.:	US 2002-87080	A1	20020227	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-284555P	20010417 (60)
	US 2001-281149P	20010402 (60)
	US 2001-272206P	20010227 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
LINE COUNT: 22670
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 10 OF 15 USPATFULL on STN
TI Genes expressed in colon cancer
AB The present invention relates to a combination comprising a plurality of cDNAs which are differentially expressed in colon cancer and which may be used in their entirety or in part as to diagnose, to stage to treat or to monitor the progression or treatment of colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:106194 USPATFULL
TITLE: Genes expressed in colon cancer
INVENTOR(S): Lasek, Amy K.W., Oakland, CA, UNITED STATES
Sornasse, Thierry, Mountain View, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003073105	A1	20030417	
APPLICATION INFO.:	US 2002-158646	A1	20020529	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-295239P	20010531 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LEGAL DEPARTMENT, INCYTE GENOMICS, INC., 3160 PORTER DRIVE, PALO ALTO, CA, 94304
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 4837
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 11 OF 15 USPATFULL on STN
TI Non-genetic based protein disease markers
AB Protein disease markers for obesity, osteoporosis, diabetes, osteoarthritis and hypertension are disclosed. These markers are not inherited or of genetic origin as they were not found in identical twins of the affected individual. Methods and uses for diagnostic, therapeutic and drug discovery are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:141506 USPATFULL
TITLE: Non-genetic based protein disease markers
INVENTOR(S): Myers, Timothy G., Kensington, MD, UNITED STATES
Pieper, Rembert, Washington, DC, UNITED STATES
Taylor, John, JR., Clayton, NC, UNITED STATES
Steiner, Sandra, Gaithersburg, MD, UNITED STATES
Anderson, N. Leigh, Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002072492	A1	20020613

APPLICATION INFO.: US 2001-886271 A1 20010622 (9)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-660242, filed on 12 Sep 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Dean H. Nakamura, Roylance Abrams Berdo & Goodman, 1300 19th Street, N.W., Washington, DC, 20036
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 1425
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 12 OF 15 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Novel combination of cDNAs which are differentially expressed in colon cancer, useful for detecting differential expression of one or more cDNAs in a sample containing nucleic acid samples.
AN AAD59167 cDNA DGENE
AB The present invention relates to combination of cDNAs which are differentially expressed in colon cancer. The invention is useful for producing and purifying antibody, utilized as markers for treatment efficacy against colon cancer. The invention is also useful for gene therapy. The present sequence is human **SNC73** protein (**SNC73**) cDNA
ACCESSION NUMBER: AAD59167 cDNA DGENE
TITLE: Novel combination of cDNAs which are differentially expressed in colon cancer, useful for detecting differential expression of one or more cDNAs in a sample containing nucleic acid samples.
INVENTOR: Lasek A K W; Sornasse T
PATENT ASSIGNEE: (LASE-I)LASEK A K W.
(SORN-I) SORNASSE T.
PATENT INFO: US 2003073105 A1 20030417 88p
APPLICATION INFO: US 2002-158646 20020529
PRIORITY INFO: US 2001-295239P 20010531
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2003-605964 [57]
DESCRIPTION: Human **SNC73** protein (**SNC73**) cDNA.

L4 ANSWER 13 OF 15 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Diagnosing and monitoring prostate disorders, by analysis of 26 gene transcripts that exhibit aberrant expression levels in prostate disorder tissues, and provides a means of early diagnosis -
AN AAD07360 DNA DGENE
AB The patent discloses a method for diagnosing, prognosing or monitoring a prostate disorder which involves the analysis of 26 gene transcripts (referred as markers) that exhibit aberrant expression levels in prostate disorder tissues and provides a means of early diagnosis. This method is useful for diagnosing, prognosing or monitoring a prostate disorder. It also provides a means of distinguishing prostate cancer from benign prostatic hyperplasia (BPH) and for identifying potential anti-prostate disorder therapeutic compounds. The present sequence is a human DNA encoding **SNC73** protein (referred as marker 11). The **SNC73** sequence is identified as an mRNA downregulated in colorectal cancer.
ACCESSION NUMBER: AAD07360 DNA DGENE
TITLE: Diagnosing and monitoring prostate disorders, by analysis of 26 gene transcripts that exhibit aberrant expression levels in prostate disorder tissues, and provides a means of early diagnosis -
INVENTOR: Bull J H; Ellison G; Paskins L D
PATENT ASSIGNEE: (ASTR)ASTRAZENECA AB.
(ASTR) ASTRAZENECA UK LTD.

PATENT INFO: WO 2001036674 A2 20010525 69p
APPLICATION INFO: WO 2000-GB4267 20001108
PRIORITY INFO: GB 1999-26805 19991113
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-343837 [36]
DESCRIPTION: Human DNA encoding **SNC73** protein (marker 11).

L4 ANSWER 14 OF 15 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Expression of a novel immunoglobulin gene **SNC73** in human cancer and non-cancerous tissues.
AB Aim: To investigate the expression of immunoglobulin gene **SNC73** in malignant tumors and non-cancerous normal tissues. Methods: Expression level of **SNC73** in tumors and non- cancerous tissues from the same patient was determined by reverse transcription polymerase chain reaction and enzyme-linked immunosorbent assay (RT-PCR-ELISA) in 90 cases of malignant tumors, including colorectal cancer, gastric cancer, breast cancer, lung cancer and liver cancer. Analysis on the correlation of **SNC73** expression with sex, age, site, grade of differentiation, depth of invasion, and metastases in colorectal cancer patients was made. Results: Expression level of **SNC73** in non-cancerous colorectal mucosa and colorectal cancerous tissues was 1.234±0.842 and 0.737±0.731, respectively ($P<0.01$), with the mean ratio of 7.134±14.092 (range, 0.36-59.54). Expression of **SNC73** showed no significant difference among gastric cancer, breast cancer, lung cancer and liver cancer when compared with non-cancerous tissues ($P>0.05$). No correlation was found between **SNC73** expression level and various clinicopathological factors, including sex, age, site, grade of differentiation, depth of invasion and metastases of CRC patients. Conclusion: Down-regulation of **SNC73** expression may be a relatively specific phenomenon in colorectal cancer. **SNC73** is a potential genetic marker for the carcinogenesis of colorectal cancer. The relationship of **SNC73** expression and carcinogenesis of colorectal cancer merits further study.
ACCESSION NUMBER: 2003228655 EMBASE
TITLE: Expression of a novel immunoglobulin gene **SNC73** in human cancer and non-cancerous tissues.
AUTHOR: Hu J.-B.; Zheng S.; Deng Y.-C.
CORPORATE SOURCE: S. Zheng, Cancer Institute, Zhejiang University, Hangzhou 310009, Zhejiang Province, China. zhengshu@mail.hz.zj.cn
SOURCE: World Journal of Gastroenterology, (15 May 2003) 9/5 (1054-1057).
Refs: 34
ISSN: 1007-9327 CODEN: WJGAF2
COUNTRY: China
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 022 Human Genetics
016 Cancer
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

L4 ANSWER 15 OF 15 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Expression of a novel immunoglobulin gene **SNC73** in human cancer and its significance.
AB Objective To investigate the expression of a new immunoglobulin gene **SNC73** in malignant tumor and normal tissue and its significance. Methods Expression level of **SNC73** in tumors and normal tissues was determined by reverse transcription-polymerase chain reaction and enzyme-linked immunosorbent assay (RT-PCR-ELISA) in 90 malignant tumors, including colorectal cancer, gastric cancer, breast cancer, lung cancer and liver cancer. Analysis on relation of **SNC73** expression with

age, sex, site, differentiation grade, depth of invasion and metastasis of colorectal cancer was made to assess the clinical significance of **SNC73**. Results Mean ratio of **SNC73** expression level in normal mucosa and colorectal cancer tissue was 7.134 ($P < 0.01$). Expression of **SNC73** showed no significant difference among gastric cancer, breast cancer, lung cancer or liver cancer as compared with the control normal tissues ($P > 0.05$). Conclusion Down-regulation of **SNC73** expression is a relatively specific phenomenon in colorectal cancer for which development **SNC73** may be a potential genetic marker. The study on relationship of **SNC73** expression with development of colorectal cancer is promising.

ACCESSION NUMBER: 2002:296037 BIOSIS
DOCUMENT NUMBER: PREV200200296037
TITLE: Expression of a novel immunoglobulin gene **SNC73** in human cancer and its significance.
AUTHOR(S): Hu Jianbin [Reprint author]; Deng Yongchuan [Reprint author]; Zheng Shu [Reprint author]
CORPORATE SOURCE: Cancer Institute, Zhejiang University, Hangzhou, 310009, China
SOURCE: Zhonghua Zhongliu Zazhi, (January, 2002) Vol. 24, No. 1, pp. 38-40. print.
CODEN: CCLCDY. ISSN: 0253-3766.
DOCUMENT TYPE: Article
LANGUAGE: Chinese
ENTRY DATE: Entered STN: 15 May 2002
Last Updated on STN: 15 May 2002

=> d l5 ti abs ibib tot

L5 ANSWER 1 OF 4 USPATFULL on STN
TI Gene sequence variations with utility in determining the treatment of disease, in genes relating to drug processing
AB Methods for identifying and utilizing variances in genes relating to efficacy and safety of medical therapy and other aspects of medical therapy are described, including methods for selecting an effective treatment.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:221287 USPATFULL
TITLE: Gene sequence variations with utility in determining the treatment of disease, in genes relating to drug processing
INVENTOR(S): Stanton, Vincent P., JR., Belmont, MA, UNITED STATES
PATENT ASSIGNEE(S): Variagenics, Inc., a Delaware corporation (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004171056	A1	20040902
APPLICATION INFO.:	US 2004-798873	A1	20040311 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-648123, filed on 25 Aug 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-590783, filed on 8 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-501955, filed on 10 Feb 2000, ABANDONED Continuation-in-part of Ser. No. WO 2000-US1392, filed on 20 Jan 2000, PENDING Continuation-in-part of Ser. No. US 1999-451252, filed on 29 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-427835, filed on 26 Oct 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-414330, filed on 6 Oct 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-389993, filed on 3 Sep 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-370841, filed

on 9 Aug 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-300747, filed on 26 Apr 1999, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-121047P	19990222 (60)
	US 1999-131334P	19990426 (60)
	US 1999-131191P	19990426 (60)
	US 1999-139440P	19990615 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
LINE COUNT: 11893
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 2 OF 4 USPATFULL on STN
TI Tumor necrosis factor receptor 2
AB The present disclosure describes the use of genetic variance information for genes involved in inflammatory or immunologic disease, disorder, or dysfunction. The variance information is indicative of the expected response of a patient to a method of treatment. Methods of determining relevant variance information and additional methods of using such variance information are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:4504 USPATFULL
TITLE: Tumor necrosis factor receptor 2
INVENTOR(S): Stanton, Jr., Vincent P., Belmont, MA, United States
PATENT ASSIGNEE(S): Nuvelo, Inc., Sunnyvale, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6673908	B1	20040106
APPLICATION INFO.:	US 2001-968455		20011001 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-649035, filed on 25 Aug 2000 Continuation-in-part of Ser. No. US 2000-590749, filed on 8 Jun 2000, now abandoned Continuation-in-part of Ser. No. US 2000-495780, filed on 1 Feb 2000, now abandoned Continuation-in-part of Ser. No. US 2000-492712, filed on 27 Jan 2000, now abandoned Continuation-in-part of Ser. No. WO 2000-US1392, filed on 20 Jan 2000 Continuation-in-part of Ser. No. US 968455 Continuation-in-part of Ser. No. US 1999-451252, filed on 29 Nov 1999, now abandoned Continuation-in-part of Ser. No. US 1999-427835, filed on 26 Oct 1999, now abandoned Continuation-in-part of Ser. No. US 1999-414330, filed on 6 Oct 1999, now abandoned Continuation-in-part of Ser. No. US 1999-389993, filed on 3 Sep 1999, now abandoned Continuation-in-part of Ser. No. US 1999-370841, filed on 9 Aug 1999, now abandoned Continuation-in-part of Ser. No. US 1999-300747, filed on 26 Apr 1999, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-131334P	19990426 (60)
	US 1999-131191P	19990426 (60)
	US 1999-121047P	19990222 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Benzion, Gary
ASSISTANT EXAMINER: Chakrabarti, Arun Kr.
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 17463
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 3 OF 4 USPATFULL on STN
TI Non-genetic based protein disease markers
AB Protein disease markers for obesity, osteoporosis, diabetes, osteoarthritis and **hypertension** are disclosed. These markers are not inherited or of genetic origin as they were not found in identical twins of the affected individual. Methods and uses for diagnostic, therapeutic and drug discovery are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:141506 USPATFULL
TITLE: Non-genetic based protein disease markers
INVENTOR(S): Myers, Timothy G., Kensington, MD, UNITED STATES
Pieper, Rembert, Washington, DC, UNITED STATES
Taylor, John, JR., Clayton, NC, UNITED STATES
Steiner, Sandra, Gaithersburg, MD, UNITED STATES
Anderson, N. Leigh, Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002072492	A1	20020613
APPLICATION INFO.:	US 2001-886271	A1	20010622 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-660242, filed on 12 Sep 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Dean H. Nakamura, Roylance Abrams Berdo & Goodman, 1300 19th Street, N.W., Washington, DC, 20036
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 1425
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 4 OF 4 USPATFULL on STN
TI Nucleic acids, proteins and antibodies
AB This invention relates to newly identified tissue specific cancer associated polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cancer antigens," and to the complete gene sequences associated therewith and to the expression products thereof, as well as the use of such tissue specific cancer antigens for detection, prevention and treatment of tissue specific disorders, particularly the presense of cancer. This invention relates to the cancer antigens as well as vectors, host cells, antibodies directed to cancer antigens and recombinant and synthetic methods for producing the same. Also provided are diagnostic methods for diagnosing and treating, preventing and/or prognosing tissue specific disorders, including cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of cancer antigens of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and/or function of the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:99407 USPATFULL
TITLE: Nucleic acids, proteins and antibodies
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002052308	A1	20020502
APPLICATION INFO.:	US 2001-925301	A1	20010810 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. WO 2000-US5882, filed on 8 Mar 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-124270P	19990312 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
LINE COUNT: 30577
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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NEWS	4	AUG 02	IFIPAT/IFIUDB/IFICDB reloaded with new search and display fields
NEWS	5	AUG 02	CAplus and CA patent records enhanced with European and Japan Patent Office Classifications
NEWS	6	AUG 02	The Analysis Edition of STN Express with Discover! (Version 7.01 for Windows) now available
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NEWS	9	SEP 01	INPADOC: New family current-awareness alert (SDI) available
NEWS	10	SEP 01	New pricing for the Save Answers for SciFinder Wizard within STN Express with Discover!
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NEWS	12	SEP 14	STN Patent Forum to be held October 13, 2004, in Iselin, NJ
NEWS	13	SEP 27	STANDARDS will no longer be available on STN
NEWS	14	SEP 27	SWETSCAN will no longer be available on STN

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NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

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FILE 'HOME' ENTERED AT 13:31:35 ON 30 SEP 2004

=> file medline,

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 13:31:41 ON 30 SEP 2004

FILE LAST UPDATED: 29 SEP 2004 (20040929/UP). FILE COVERS 1951 TO DATE.

On February 29, 2004, the 2004 MeSH terms were loaded. See HELP RLOAD for details. OLDMEDLINE now back to 1951.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2004 vocabulary. See http://www.nlm.nih.gov/mesh/ and http://www.nlm.nih.gov/pubs/techbull/nd03/nd03_mesh.html for a description of changes.

This file contains CAS Registry Numbers for easy and accurate substance identification.

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=> s obesity and disease marker
         73480 OBESITY
       1598365 DISEASE
        119367 MARKER
           191 DISEASE MARKER
                 (DISEASE(W)MARKER)
L1           1 OBESITY AND DISEASE MARKER

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L1 ANSWER 1 OF 1 MEDLINE on STN

TI Coronary heart disease risk prediction in the Atherosclerosis Risk in Communities (ARIC) study.

AB Risk prediction functions for incident coronary heart disease (CHD) were estimated using data from the Atherosclerosis Risk in Communities (ARIC) Study, a prospective study of CHD in 15,792 persons recruited in 1987-1989 from four U.S. communities, with follow-up through 1998. Predictivity of which individuals had incident CHD was assessed by increase in area under ROC curves resulting from adding nontraditional risk factors and markers of subclinical disease to a basic model containing only traditional risk factors. We also assessed the increase in population attributable risk. The additional factors were body mass index; waist-hip ratio; sport activity index; forced expiratory volume; plasma fibrinogen, factor VIII, von Willebrand factor, and Lp(a); heart rate; Keys score; pack-years smoking; and subclinical **disease marker** carotid intima-media thickness. These factors substantially improved prediction of future CHD for men, less for women, and also increased attributable risks.

ACCESSION NUMBER: 2003445840 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14505774
TITLE: Coronary heart disease risk prediction in the Atherosclerosis Risk in Communities (ARIC) study.
AUTHOR: Chambless Lloyd E; Folsom Aaron R; Sharrett A Richey; Sorlie Paul; Couper David; Szklo Moyses; Nieto F Javier
CORPORATE SOURCE: Department of Biostatistics, University of North Carolina, CB #8300, 137 East Franklin Street, Suite 400, Bank of America Center, Chapel Hill, NC 27514-4145, USA..
wchambless@unc.edu
CONTRACT NUMBER: N01-HC-55015 (NHLBI)
N01-HC-55016 (NHLBI)
N01-HC-55018 (NHLBI)
N01-HC-55019 (NHLBI)
N01-HC-55020 (NHLBI)
N01-HC-55021 (NHLBI)
N01-HC-55022 (NHLBI)
SOURCE: Journal of clinical epidemiology, (2003 Sep) 56 (9) 880-90.
Journal code: 8801383. ISSN: 0895-4356.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 20030925
Last Updated on STN: 20031218
Entered Medline: 20031204

=> s osteoporosis and disease marker
31003 OSTEOPOROSIS
1598365 DISEASE
119367 MARKER
191 DISEASE MARKER
(DISEASE(W)MARKER)
L2 0 OSTEOPOROSIS AND DISEASE MARKER

=> s l1 and protein marker
1311316 PROTEIN
119367 MARKER
225 PROTEIN MARKER
(PROTEIN(W)MARKER)
L3 0 L1 AND PROTEIN MARKER

=> s diabetes and disease marker
206298 DIABETES
1598365 DISEASE
119367 MARKER
191 DISEASE MARKER
(DISEASE(W)MARKER)
L4 19 DIABETES AND DISEASE MARKER

=> s l4 and protein marker
1311316 PROTEIN
119367 MARKER
225 PROTEIN MARKER
(PROTEIN(W)MARKER)
L5 0 L4 AND PROTEIN MARKER

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L4 ANSWER 1 OF 19 MEDLINE on STN
TI The emerging value of P-selectin as a **disease marker**.
AB Activated platelets are key components in many arterial disorders. P-selectin is an activation-dependent platelet receptor, which is also identified in endothelial cells. Together with E- and L-selectin it constitutes the selectin family. These transmembrane proteins have continued to attract great interest as they support rapid and reversible cell adhesion in flow systems and thus play an essential role in multicellular interactions during thrombosis and inflammation. Similarly to other lectins, selectins bind to different glycoconjugates with varying affinities. Protein ligands, equipped with the appropriate carbohydrate and sulfate moieties for P-selectin binding, have been identified in normal peripheral blood leukocytes and several non-hematopoietic organs, as well as on cancer cells. For diagnostic purposes, P-selectin can readily be detected on the platelet surface by flow cytometry and by ELISA as a soluble ligand in the plasma. Along with other markers, these data can be used in the assessment of platelet activation status. Such results bear clinical significance since P-selectin has been implicated in the pathogenesis of wide-spread disorders including coronary artery disease, stroke, **diabetes** and malignancy.
ACCESSION NUMBER: 2004301573 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15202782
TITLE: The emerging value of P-selectin as a **disease marker**.

AUTHOR: Kappelmayer Janos; Nagy Bela Jr; Miszti-Blasius Kornel; Hevessy Zsuzsa; Setiadi Hendra
CORPORATE SOURCE: Department of Clinical Biochemistry and Molecular Pathology, Medical and Health Science Center, University of Debrecen, Debrecen, Hungary.. kappelmayer@jaguar.dote.hu
SOURCE: Clinical chemistry and laboratory medicine : CCLM / FESCC, (2004 May) 42 (5) 475-86.
Journal code: 9806306. ISSN: 1434-6621.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20040624
Last Updated on STN: 20040624

L4 ANSWER 2 OF 19 MEDLINE on STN
TI Rapid determination of acetone in human plasma by gas chromatography-mass spectrometry and solid-phase microextraction with on-fiber derivatization.
AB Acetone is an important volatile **disease marker**. Due to its nature of activity and volatility, it is a difficult task to measure the concentration of acetone in biological samples with accuracy. In this paper, we developed a novel method for determination of trace amount acetone in human plasma by solid-phase microextraction technique with on-fiber derivatization. In this method, the poly(dimethylsiloxane)/divinylbenzene (PDMS/DVB) fiber was used and O-2,3,4,5,6-(pentafluorobenzyl) hydroxylamine hydrochloride (PFBHA) was first loaded on the fiber. Acetone in plasma sample was agitated into headspace and extracted by solid-phase microextraction (SPME) fiber and subsequently derivatized with PFBHA on the fiber. Acetone oxime was analyzed by gas chromatography-mass spectrometry (GC-MS). Quantitative analysis of acetone in plasma was carried out by using external standard method. The SPME conditions (extraction temperature and time) and the method validation were studied. The present method was tested by determination of acetone in **diabetes** plasma and normal plasma. Acetone concentration in **diabetes** plasma was found to be higher than 1.8mM, while in normal plasma was lower than 0.017 mM. The results show that the present method is a potential tool for diagnosis of **diabetes**.
ACCESSION NUMBER: 2004237499 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15135095
TITLE: Rapid determination of acetone in human plasma by gas chromatography-mass spectrometry and solid-phase microextraction with on-fiber derivatization.
AUTHOR: Deng Chunhui; Zhang Wei; Zhang Jie; Zhang Xiangmin
CORPORATE SOURCE: Department of Chemistry, Fudan University, Shanghai 200433, PR China.
SOURCE: Journal of chromatography. B, Analytical technologies in the biomedical and life sciences, (2004 Jun 15) 805 (2) 235-40.
Journal code: 101139554. ISSN: 1570-0232.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20040512
Last Updated on STN: 20040714

L4 ANSWER 3 OF 19 MEDLINE on STN
TI Coronary heart disease risk prediction in the Atherosclerosis Risk in Communities (ARIC) study.
AB Risk prediction functions for incident coronary heart disease (CHD) were estimated using data from the Atherosclerosis Risk in Communities (ARIC) Study, a prospective study of CHD in 15,792 persons recruited in 1987-1989 from four U.S. communities, with follow-up through 1998. Predictivity of

which individuals had incident CHD was assessed by increase in area under ROC curves resulting from adding nontraditional risk factors and markers of subclinical disease to a basic model containing only traditional risk factors. We also assessed the increase in population attributable risk. The additional factors were body mass index; waist-hip ratio; sport activity index; forced expiratory volume; plasma fibrinogen, factor VIII, von Willebrand factor, and Lp(a); heart rate; Keys score; pack-years smoking; and subclinical **disease marker** carotid intima-media thickness. These factors substantially improved prediction of future CHD for men, less for women, and also increased attributable risks.

ACCESSION NUMBER: 2003445840 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14505774
TITLE: Coronary heart disease risk prediction in the Atherosclerosis Risk in Communities (ARIC) study.
AUTHOR: Chambless Lloyd E; Folsom Aaron R; Sharrett A Richey; Sorlie Paul; Couper David; Szklo Moyses; Nieto F Javier
CORPORATE SOURCE: Department of Biostatistics, University of North Carolina, CB #8300, 137 East Franklin Street, Suite 400, Bank of America Center, Chapel Hill, NC 27514-4145, USA.. wchambless@unc.edu
CONTRACT NUMBER: N01-HC-55015 (NHLBI)
N01-HC-55016 (NHLBI)
N01-HC-55018 (NHLBI)
N01-HC-55019 (NHLBI)
N01-HC-55020 (NHLBI)
N01-HC-55021 (NHLBI)
N01-HC-55022 (NHLBI)
SOURCE: Journal of clinical epidemiology, (2003 Sep) 56 (9) 880-90. Journal code: 8801383. ISSN: 0895-4356.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 20030925
Last Updated on STN: 20031218
Entered Medline: 20031204

L4 ANSWER 4 OF 19 MEDLINE on STN
TI Testing for population subdivision and association in four case-control studies.
AB Population structure has been presumed to cause many of the unreplicated **disease-marker** associations reported in the literature, yet few actual case-control studies have been evaluated for the presence of structure. Here, we examine four moderate case-control samples, comprising 3,472 individuals, to determine if detectable population subdivision is present. The four population samples include: 500 U.S. whites and 236 African Americans with hypertension; and 500 U.S. whites and 500 Polish whites with type 2 **diabetes**, all with matched control subjects. Both **diabetes** populations were typed for the PPARg Pro12Ala polymorphism, to replicate this well-supported association (Altshuler et al. 2000). In each of the four samples, we tested for structure, using the sum of the case-control allele frequency chi(2) statistics for 9 STR and 35 SNP markers (Pritchard and Rosenberg 1999). We found weak evidence for population structure in the African American sample only, but further refinement of the sample, to include only individuals with U.S.-born parents and grandparents, eliminated the stratification. Our examples provide insight into the factors affecting the replication of association studies and suggest that carefully matched, moderate-sized case-control samples in cosmopolitan U.S. and European populations are unlikely to contain levels of structure that would result in significantly inflated numbers of false-positive associations. We explore the role that extreme differences in power among studies, due to

sample size and risk-allele frequency differences, may play in the replication problem.

ACCESSION NUMBER: 2002365873 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12096349
TITLE: Testing for population subdivision and association in four case-control studies.
COMMENT: Comment in: Am J Hum Genet. 2002 Dec;71(6):1478-80. PubMed ID: 12515254
AUTHOR: Ardlie Kristin G; Lunetta Kathryn L; Seielstad Mark
CORPORATE SOURCE: Genomics Collaborative, 99 Erie Street, Cambridge, MA, 02139, USA.. kardlie@genomicsinc.com
SOURCE: American journal of human genetics, (2002 Aug) 71 (2) 304-11.
Journal code: 0370475. ISSN: 0002-9297.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20020712
Last Updated on STN: 20030124
Entered Medline: 20020820

L4 ANSWER 5 OF 19 MEDLINE on STN
TI The natural history of renal disease in Australian Aborigines. Part 2. Albuminuria predicts natural death and renal failure.
AB BACKGROUND: The purpose of this study was to describe the relationship of albuminuria and glomerular filtration rate (GFR) with natural death and renal failure in an Australian Aboriginal community with high rates of renal disease. METHODS: Study subjects were 825 adults (18+ years, mean 33.6 years) or 88% of adults in a remote community who participated in a health screening program offered between 1990 and 1997. The urinary albumin:creatinine ratio (ACR; g/mol) was used as the renal **disease marker**. Participants were followed for 1.0 to 9.8 years (mean 5.8 years) until renal failure, death, the start of systematic antihypertensive/renal-protective treatment or June 30, 2000. RESULTS: Sixty-five people reached a terminal end point of renal failure or natural death. Sixteen people developed terminal renal failure, all of whom had an ACR of 34+ at baseline exam. There were 49 other natural deaths, which were also strongly correlated with increasing ACR and decreasing GFR over a wide range. This was observed in people without **diabetes** and in people with normal and elevated blood pressures. It applied to deaths associated with cardiovascular disease and to deaths without an assigned primary or underlying cardiovascular or renal cause. With adjustment for age, the association with death was more robust with ACR than GFR. When compared with people with an ACR <3.4, the hazard ratio (HR; 95% CI) for nonrenal natural death of persons with an ACR 3.4 to 33 was 3.0 (1.1 to 8.4), with an ACR 34 to 99, it was 5.4 (1.8 to 15.9), and with an ACR 100+, it was 6.5 (2.0 to 21). Regression equations predicted that each tenfold increase in the ACR was associated with a 3.7-fold increase in all-cause natural death: a> 400-fold increase in renal deaths, a 4-fold increase in cardiovascular deaths, and a 2.2-fold increase in nonrenal noncardiovascular deaths. Eighty-four percent of all-cause natural death was associated with pathologic albuminuria. CONCLUSION: All renal failure develops out of a background of persistent albuminuria in this population. More important, albuminuria and, inversely, GFR are powerful markers of risk for nonrenal natural death, including, but not restricted to, cardiovascular deaths. Most of the risk for premature death can be assessed by a simple urine test, and interventions that prevent development and progression of albuminuria and loss of GFR should not only prevent renal insufficiency, but powerfully reduce mortality from natural causes as well.

ACCESSION NUMBER: 2001362796 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11422758

TITLE: The natural history of renal disease in Australian Aborigines. Part 2. Albuminuria predicts natural death and renal failure.
AUTHOR: Hoy W E; Wang Z; VanBuynder P; Baker P R; McDonald S M; Mathews J D
CORPORATE SOURCE: Menzies School of Health Research, Darwin, Northern Territory, Australia.
SOURCE: Kidney international, (2001 Jul) 60 (1) 249-56. Journal code: 0323470. ISSN: 0085-2538.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010903
Last Updated on STN: 20010903
Entered Medline: 20010830

L4 ANSWER 6 OF 19 MEDLINE on STN
TI Antibodies to SOX13 (ICA12) are associated with type 1 **diabetes**.
AB SOX13 is an islet cell autoantigen (ICA12), identified by antibody screening of an islet cDNA library, using sera from patients with Type 1 **diabetes**. We ascertained the frequency of antibody reactivity to SOX13 and compared it with other Type 1 **diabetes** autoantibody reactivities. Antibodies were measured by radioimmunoprecipitation (RIP) using (35) S labelled SOX13 expressed in rabbit reticulocyte lysate. Sera from 109 subjects with Type 1 **diabetes**, 29 with Type 2 **diabetes**, 144 with other autoimmune diseases and from 201 controls were tested for anti-SOX13, and results were compared with the frequency of antibodies to glutamic acid decarboxylase (anti-GAD), islet cell antigen 512 (anti-ICA512) and islet cell cytoplasm (ICA). Anti-SOX13 were detected in 20 (18.3%) of 109 subjects with Type 1 **diabetes**, and more frequently in adults than in children (29% vs 10%). Anti-SOX13 usually occurred with anti-GAD but rarely with anti-ICA512. Seven sera positive for anti-SOX13 did not react with either GAD, ICA512 or islet cell cytoplasm indicating that anti-SOX13 represented a distinct population of antibodies. Reactivity to SOX13 represents a further autoantibody response in adults with Type 1 **diabetes** and may provide a useful **disease marker** in subjects in whom other autoantibody tests are negative.
ACCESSION NUMBER: 2001166084 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11264788
TITLE: Antibodies to SOX13 (ICA12) are associated with type 1 **diabetes**.
AUTHOR: Kasimiotis H; Fida S; Rowley M J; Mackay I R; Zimmet P Z; Gleason S; Rabin D U; Myers M A
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Monash University, Wellington Road Clayton, 3168 Australia.
SOURCE: Autoimmunity, (2001) 33 (2) 95-101. Journal code: 8900070. ISSN: 0891-6934.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200109
ENTRY DATE: Entered STN: 20010917
Last Updated on STN: 20010917
Entered Medline: 20010913

L4 ANSWER 7 OF 19 MEDLINE on STN
TI The multidimensional nature of renal disease: rates and associations of albuminuria in an Australian Aboriginal community.
AB BACKGROUND: An epidemic of end-stage renal disease (ESRD) is accompanying the rising rates of hypertension, type 2 **diabetes** and

cardiovascular disease among Aborigines in the Northern Territory of Australia. Incidence rates are now 21 times those of nonAboriginal Australians and are doubling every four years. We describe the rates and associations of renal disease in one remote community, which has a current ESRD incidence of 2700 per million, and cardiovascular mortality among the highest in Australia. METHODS: Between 1992 and 1995 a community-wide screening program was conducted, in which the urinary albumin/creatinine ratio (ACR) was used as the chief renal **disease marker** . More than 90% of the population ages five and older participated. RESULTS: Albuminuria was evident in early childhood and increased dramatically with age; 26% of adults had microalbuminuria and 24% had overt albuminuria. All renal failure developed out of a background of overt albuminuria. ACR was significantly correlated with the presence of scabies at screening, with a history of poststreptococcal glomerulonephritis, which is epidemic and endemic in the community, with increasing body wt, blood pressure, glucose, insulin and lipid levels, and with evidence of heavy drinking. ACR was also significantly and inversely correlated with birth weight. As a result of its association with deteriorating hemodynamic and metabolic profiles, increasing ACR was also correlated with increasing cardiovascular risk score. Direct observations showed, and multivariate models predicted, progressive amplification of ACR when multiple risk factors were present simultaneously. Albuminuria also clustered in families. Conclusion: Renal disease in this population is multifactorial, with risk factors related to whole-of-life nutrition, metabolic and hemodynamic profiles, infections, health behaviors, and possibly a family predisposition. Its relationship to low birth weight, and its associations with deteriorating metabolic and hemodynamic profiles, suggest that renal disease is, in part, a component of Syndrome X, which explains the simultaneous increase in metabolic, cardiovascular and renal disease in Aboriginal people. The family clustering might have both environmental and genetic causes, and is under further investigation. Most of the identified risk factors arise out of poverty, disadvantage and accelerated lifestyle change, and the current epidemic can be explained by the confluence of many risk factors in the last few decades. The introduction of effective and sustained programs to address social, economic and educational inequities in all Aboriginal communities, and of screening and renal- and cardiovascular-protective treatment programs for those already afflicted are matters of great urgency.

ACCESSION NUMBER: 1998444650 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9767547
TITLE: The multidimensional nature of renal disease: rates and associations of albuminuria in an Australian Aboriginal community.
AUTHOR: Hoy W E; Mathews J D; McCredie D A; Pugsley D J; Hayhurst B G; Rees M; Kile E; Walker K A; Wang Z
CORPORATE SOURCE: Menzies School of Health Research, Darwin, Northern Territory, Australia.. wendy@menzies.su.edu.au
SOURCE: Kidney international, (1998 Oct) 54 (4) 1296-304. Journal code: 0323470. ISSN: 0085-2538.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981231

L4 ANSWER 8 OF 19 MEDLINE on STN

TI A likelihood ratio test for detecting patterns of **disease-marker** association.

AB A likelihood ratio test of **disease-marker** association is proposed, based on the observation of marker alleles transmitted from parents to affected children. The proposed association test has the

advantage of identifying the population pattern of **disease-marker** association, differentiating between marker alleles that are positively and negatively associated with the disease. The power of the test for detecting association is evaluated and compared with three existing multi-allelic tests for some specific **disease-marker** association patterns. The power of the parametric tests depends crucially on the pattern of **disease-marker** association. An over-parameterised association model is less detrimental in terms of power than an under parameterised model.

ACCESSION NUMBER: 1998032490 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9365786
TITLE: A likelihood ratio test for detecting patterns of **disease-marker** association.
AUTHOR: Morris A P; Whittaker J C; Curnow R N
CORPORATE SOURCE: University of Reading, Department of Applied Statistics.. A.P.Morris@reading.ac.uk
SOURCE: Annals of human genetics, (1997 Jul) 61 (Pt 4) 335-50. Journal code: 0416661. ISSN: 0003-4800.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971210

L4 ANSWER 9 OF 19 MEDLINE on STN
TI Antibodies to bovine serum albumin (BSA) in type 1 **diabetes** and other autoimmune disorders.
AB Clinical and experimental studies have delineated a link between dietary cow milk protein and the development of insulin-dependent **diabetes** mellitus (IDDM), and bovine serum albumin (BSA) was proposed as one candidate mediator of this effect. The demonstration of anti-BSA antibodies in new onset type 1-diabetic children from Finland initiated a controversial debate on the utility of BSA antibodies as a **disease marker** and on the role of BSA in IDDM. Here we analyzed BSA antibodies in newly diagnosed type 1-diabetic patients and their first degree relatives, patients with other autoimmune diseases, and children with Down's syndrome from Germany. Blinded serum samples (n = 308) were screened for IgG anti-BSA antibodies by particle concentration fluoroimmunoassay (PCFIA). The prevalence of elevated BSA antibodies in newly diagnosed type 1-diabetic patients was low (11%), although mean BSA antibody levels were significantly increased in diabetic patients as compared to controls (1.94 +/- 1.51 vs. 0.97 +/- 0.93 kFU, $p < 0.0007$). Mean BSA antibody levels were also increased in ICA+ and/or IAA+ first degree relatives (1.32 +/- 0.43, $p < 0.002$) and in children with Down's syndrome (3.01 +/- 1.93, $p < 0.0007$), but not in the other autoimmune disorders tested. The low prevalence of elevated anti-BSA levels in IDDM patients limits the clinical usefulness of this immune marker. We conclude that current anti-BSA assays do not substantially contribute to the prediction and diagnosis of IDDM.

ACCESSION NUMBER: 97283862 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9137938
TITLE: Antibodies to bovine serum albumin (BSA) in type 1 **diabetes** and other autoimmune disorders.
COMMENT: Comment in: Exp Clin Endocrinol Diabetes. 1997;105(2):83-5. PubMed ID: 9137937
AUTHOR: Fuchtenbusch M; Karges W; Standl E; Dosch H M; Ziegler A G III.
CORPORATE SOURCE: Medical Department, Schwabing City Hospital, Munich, Germany.
SOURCE: Experimental and clinical endocrinology & diabetes : official journal, German Society of Endocrinology [and] German Diabetes Association, (1997) 105 (2) 86-91.

	Journal code: 9505926. ISSN: 0947-7349.
PUB. COUNTRY:	GERMANY: Germany, Federal Republic of
DOCUMENT TYPE:	(CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	199707
ENTRY DATE:	Entered STN: 19970724 Last Updated on STN: 20021217 Entered Medline: 19970717

L4 ANSWER 10 OF 19 MEDLINE on STN

TI High prevalence of autoantibodies to glutamic acid decarboxylase in long-standing IDDM is not a marker of symptomatic autonomic neuropathy.

AB Immune reactivity to the enzyme glutamic acid decarboxylase (GAD), a pancreatic islet autoantigen, is present at the diagnosis of insulin-dependent **diabetes** mellitus (IDDM). Because GAD is also highly expressed in the nervous system, we investigated the presence of autoantibodies to the isoform GAD65 in patients with diabetic neuropathy, which is a debilitating complication of the disease. We studied 39 patients with autonomic and somatic neuropathy, 28 patients matched for age and IDDM duration, and 13 patients with a shorter duration of IDDM, all with no diabetic complications, as well as 50 recently diagnosed diabetic patients, 23 neurologic patients with idiopathic autonomic failure unrelated to IDDM, and 72 healthy subjects. An immunoprecipitation radioligand assay was used to detect anti-GAD65 autoantibodies with in vitro transcribed and translated human islet GAD65 as antigen. Autoantibodies to GAD65 were present in 56% of the diabetic patients with neuropathy, 57% of the long-duration and 69% of the short-duration diabetic control subjects, 78% of the recently diagnosed patients, and 13% of the nondiabetic neuropathic patients. Among the diabetic patients with neuropathy, there was no correlation between the presence of anti-GAD65 antibodies and the presence of autoantibodies to sympathetic ganglia, vagus nerve, or adrenal medulla structures identified by immunofluorescence. Our study shows that anti-GAD65 antibodies are present in a high proportion of patients with diabetic neuropathy but are not exclusively associated with it, rendering it unlikely that they have a role as a **disease marker** or that they are pathogenetic.(ABSTRACT TRUNCATED AT 250 WORDS)

ACCESSION NUMBER:	94350155 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 8070615
TITLE:	High prevalence of autoantibodies to glutamic acid decarboxylase in long-standing IDDM is not a marker of symptomatic autonomic neuropathy.
AUTHOR:	Zanone M M; Petersen J S; Peakman M; Mathias C J; Watkins P J; Dyrberg T; Vergani D
CORPORATE SOURCE:	Immunology Department, King's College School of Medicine and Dentistry, London, U.K.
SOURCE:	Diabetes, (1994 Sep) 43 (9) 1146-51. Journal code: 0372763. ISSN: 0012-1797.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:	199409
ENTRY DATE:	Entered STN: 19941006 Last Updated on STN: 19941006 Entered Medline: 19940923

L4 ANSWER 11 OF 19 MEDLINE on STN

TI Serum sialic acid, a risk factor for cardiovascular disease, is increased in IDDM patients with microalbuminuria and clinical proteinuria.

AB OBJECTIVE--An elevated serum sialic acid concentration has recently been shown to be a potent cardiovascular risk factor in the general population.

Because clinical proteinuria is associated with a high frequency of cardiovascular disease, and because microalbuminuria predicts the development of renal and cardiovascular disease in **diabetes**, we investigated whether serum sialic acid levels are increased in insulin-dependent **diabetes** mellitus (IDDM) patients with microalbuminuria or clinical proteinuria. RESEARCH DESIGN AND METHODS--We studied 23 patients with IDDM who had a normal urinary albumin excretion rate, 23 patients who had microalbuminuria, and 23 patients with clinical proteinuria. The patients were matched for age, sex, duration of **diabetes**, GHb levels, and body mass index (BMI). Fasting blood samples were taken for measurement of sialic acid, cholesterol, triglyceride, creatinine, and GHb. RESULTS--Serum sialic acid was significantly higher in the microalbuminuric patients compared with the normoalbuminuric group (mean +/- SD: 1.93 +/- 0.26 vs. 1.76 +/- 0.27 mM, P < 0.01). Moreover, serum sialic acid was also significantly higher in the group with clinical proteinuria compared with the microalbuminuric patients (2.34 +/- 0.24 vs. 1.93 +/- 0.26 mM, P < 0.001). Serum sialic acid was not related independently to age, BMI, **diabetes** duration, GHb, blood pressure, serum cholesterol, triglyceride, or creatinine concentration in any of the diabetic groups. CONCLUSIONS--These observations suggest that the serum sialic acid concentration is raised in IDDM patients with both microalbuminuria and clinical proteinuria and may play a role as a cardiovascular risk factor or **disease marker** in these conditions.

ACCESSION NUMBER: 94298476 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8026286
TITLE: Serum sialic acid, a risk factor for cardiovascular disease, is increased in IDDM patients with microalbuminuria and clinical proteinuria.
AUTHOR: Crook M A; Earle K; Morocutti A; Yip J; Viberti G; Pickup J C
CORPORATE SOURCE: Division of Chemical Pathology, United Medical School, Guy's Hospital, London, United Kingdom.
SOURCE: Diabetes care, (1994 Apr) 17 (4) 305-10. Journal code: 7805975. ISSN: 0149-5992.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940818
Last Updated on STN: 19940818
Entered Medline: 19940811

L4 ANSWER 12 OF 19 MEDLINE on STN
TI No independent association between a tumor necrosis factor-alpha promotor region polymorphism and insulin-dependent **diabetes** mellitus.
AB Several studies have implicated tumor necrosis factor (TNF)-alpha in the pathogenesis of insulin-dependent **diabetes** mellitus (IDDM). In the present study we analyzed the first reported TNF-alpha gene polymorphism in relation to IDDM. We have made frequence analysis and tested in vitro lipopolysaccharide (LPS)-induced TNF-alpha secretion. A significant difference in allele frequency was observed between patients and controls (p = 0.03). However, a very strong association of the uncommon TNF2 allele was observed with the HLA-B8, -DR3 alleles. The relative risk (RR) of TNF2 was 2.2 compared to a RR of 3.1 for DR3. One reason for this difference was the identification of the TNF1 allele on the otherwise strongly IDDM-associated HLA-DR3 haplotype: DQB1*0201, DQA1*0501, DRB1*0301, TNFc2, TNFB*2, TNFa1, TNFb5, B18. Thus, the IDDM-associated TNF2 allele had no DR3-independent value as a **disease marker**. The LPS-induced TNF-alpha production by human monocytes in relation to genotypes demonstrated that TNF1/2 heterozygous individuals had higher, though not statistically significantly (p = 0.08) levels than TNF1-homozygous subjects. However,

this difference was rather small, unlikely to be of biological significance and based on the present material we cannot establish the functional importance of this polymorphism.

ACCESSION NUMBER: 94039413 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8223882
TITLE: No independent association between a tumor necrosis factor-alpha promotor region polymorphism and insulin-dependent **diabetes** mellitus.
AUTHOR: Pociot F; Wilson A G; Nerup J; Duff G W
CORPORATE SOURCE: Steno Diabetes Center, Gentofte, Denmark.
SOURCE: European journal of immunology, (1993 Nov) 23 (11) 3050-3. Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199312
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 19940117
Entered Medline: 19931214

L4 ANSWER 13 OF 19 MEDLINE on STN
TI Advanced glycosylation end products: a new **disease marker** for **diabetes** and aging.
AB Advanced glycosylation end products (AGEs) are a potentially useful marker for monitoring glycemic control, predicting the risk of **diabetes** - and aging-associated clinical complications, and monitoring the treatment of patients with micro- and macrovascular diseases, including retinopathy, atherosclerosis, nephropathy, and neuropathy. AGEs or AGE-proteins are derived from nonenzymatically glycated proteins (Amadori products) after further cross-linking with other proteins and additional rearrangement. AGE-proteins can be assayed by either radioreceptor or immunoassays in blood and tissues. No commercial kit is available at this time.

ACCESSION NUMBER: 94015658 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8410484
TITLE: Advanced glycosylation end products: a new **disease marker** for **diabetes** and aging.
AUTHOR: Wu J T
CORPORATE SOURCE: Department of Pathology, University of Utah Medical Center, Salt Lake City 84108.
SOURCE: Journal of clinical laboratory analysis, (1993) 7 (5) 252-5. Journal code: 8801384. ISSN: 0887-8013.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199311
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 19940117
Entered Medline: 19931109

L4 ANSWER 14 OF 19 MEDLINE on STN
TI HLA-DQB1 alleles and absence of Asp 57 as susceptibility factors of IDDM in Finland.
AB It has been proposed that negatively charged aspartic acid at position 57 of the HLA-DQ beta-chain determines resistance to development of insulin-dependent **diabetes** mellitus (IDDM), whereas genetic susceptibility to IDDM correlates with a neutral amino acid residue. The disease rate is very low in Oriental populations with high frequencies of Asp 57. This raises a question whether the high incidence of IDDM in Finland could be explained by the distribution of this **disease marker**. In this study, the polymerase chain reaction products of

86 diabetic patients and 115 nondiabetic control subjects were analyzed with seven sequence-specific oligonucleotide probes. Only 25.5% of the diabetic subjects were phenotyped as Asp 57+ compared to 82% of control subjects, which suggests that Asp 57 negativity is a definite risk marker for developing IDDM in Finnish patients. However, the susceptibility conferred by various non-Asp and Asp haplotypes was not equally strong: DQw8 was the most important risk marker and DQw6 the most protective one. The frequency of Asp 57+ DQw4 was similar in diabetic patients and control subjects. The highest genotype-associated relative risk was defined by DQw2/DQw8 heterozygosity (RR 91), whereas it was 13 for non-Asp homozygosity. In the control subjects, the frequency of Asp 57+ phenotypes was higher than in several white populations with lower IDDM incidence figures. We conclude that the disease risk in Finland appears to be most strongly related to specific Asp 57- alleles, although other HLA- or non-HLA-associated genes may also contribute to IDDM susceptibility in this population.

ACCESSION NUMBER: 92097856 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1756904
TITLE: HLA-DQB1 alleles and absence of Asp 57 as susceptibility factors of IDDM in Finland.
AUTHOR: Reijonen H; Ilonen J; Knip M; Akerblom H K
CORPORATE SOURCE: Department of Medical Microbiology, University of Oulu, Finland.
SOURCE: Diabetes, (1991 Dec) 40 (12) 1640-4.
Journal code: 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 19920223
Last Updated on STN: 19920223
Entered Medline: 19920203

L4 ANSWER 15 OF 19 MEDLINE on STN
TI Complement component 3 (C3) genetics and **diabetes** mellitus.
AB Complement component 3 (C3) phenotype and allele frequencies were defined in 312 patients with type-1 **diabetes** (insulin-dependent **diabetes** mellitus), 256 patients with type-2 **diabetes** (non-insulin-dependent **diabetes** mellitus), 114 apparently non-diabetic first-degree relatives of type-1 diabetics, in 10 families (29 members) with a familial history of type-1 or type-2 **diabetes** , in 181 patients with coronary heart disease and 255 subjects with arterial hypertension. 512 blood donors served as controls. All persons investigated were Europeans. There is no evidence that genes linked to C3 influence susceptibility to type-1 and type-2 **diabetes** and to their late complications as well as to atherosclerosis and essential hypertension. The distribution of apolipoprotein E phenotypes in patients and controls was likewise not significantly different. The combined evaluation of data from linked genes (C3 and apo E) could not improve the results. Deductions of C3 as a genetic **disease marker** have to be interpreted with caution.

ACCESSION NUMBER: 91273632 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2097995
TITLE: Complement component 3 (C3) genetics and **diabetes** mellitus.
AUTHOR: Krantz S; Stelter F; Lober M; Gromoll B
CORPORATE SOURCE: Institute of Biochemistry, Ernst Moritz Arndt University, Greifswald, FRG.
SOURCE: Biomedica biochimica acta, (1990) 49 (12) 1237-41.
Journal code: 8304435. ISSN: 0232-766X.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199107
ENTRY DATE: Entered STN: 19910811
Last Updated on STN: 19910811
Entered Medline: 19910725

L4 ANSWER 16 OF 19 MEDLINE on STN
TI Disease associations. Chance, artifact, or susceptibility genes?.
AB Numerous genes that might contribute to the development of **diabetes** mellitus and/or its complications have been isolated and characterized. One approach to determining whether these "candidate" genes influence susceptibility to **diabetes** is to compare the frequency of a DNA marker(s) (restriction-fragment-length polymorphism) for each gene is appropriately matched groups of patients and control subjects. The identification of a DNA-marker association would suggest that genetic variation at this gene may increase or reduce the risk of developing **diabetes**. However, the absence of an association does not necessarily imply that this gene does not contribute to the development of **diabetes**. We discuss the genetic rationale of disease association studies and the importance of sample size and **disease-marker** allele frequencies in these studies.
ACCESSION NUMBER: 89325861 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2568956
TITLE: Disease associations. Chance, artifact, or susceptibility genes?.
AUTHOR: Cox N J; Bell G I
CORPORATE SOURCE: Howard Hughes Medical Institute, University of Chicago, IL 60637.
SOURCE: Diabetes, (1989 Aug) 38 (8) 947-50. Ref: 18
Journal code: 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198909
ENTRY DATE: Entered STN: 19900309
Last Updated on STN: 19950206
Entered Medline: 19890907

L4 ANSWER 17 OF 19 MEDLINE on STN
TI Cell-mediated immunity in the aetiopathogenesis of insulin-dependent (type I) **diabetes** mellitus.
AB We have investigated lymphocyte subpopulation levels with monoclonal antibodies in newly diagnosed insulin-dependent (type I) diabetics and in unaffected siblings of type I diabetic probands with islet cell antibodies. Our data show that in newly diagnosed diabetics there is 1) a decrease in T cells with suppressor phenotype, 2) an increase of T cells with cytotoxic phenotype and 3) the presence of "activated" T cells. The latter have also been found in some unaffected siblings with islet cell antibodies. These results suggest that cellular immune alterations are present, not only at diagnosis, but also in normal but "susceptible" individuals. "Activated" T cells could be a "**disease** **marker**, but their better definition in terms of specificity should be established.
ACCESSION NUMBER: 84307492 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6236800
TITLE: Cell-mediated immunity in the aetiopathogenesis of insulin-dependent (type I) **diabetes** mellitus.
AUTHOR: Pozzilli P; Zuccarini O; Sensi M; Spencer K M; Bottazzo G
SOURCE: Biomedica biochimica acta, (1984) 43 (5) 621-5.
Journal code: 8304435. ISSN: 0232-766X.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198410
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19841019

L4 ANSWER 18 OF 19 MEDLINE on STN
TI A general model for **disease-marker** association.
AB A general model for analysing **disease-marker** associations from a random sample of patients and controls is given, assuming an arbitrary number of marker and disease susceptibility alleles. A method for testing the goodness-of-fit of various disease susceptibility models to the observed distribution of genotypes at the marker locus in patient and control samples is given. The method is demonstrated using a recently published data set on type I **diabetes**.
ACCESSION NUMBER: 83307182 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6577811
TITLE: A general model for **disease-marker** association.
AUTHOR: Risch N
CONTRACT NUMBER: KO4 HD00477 (NICHD)
MH 30906-03 (NIMH)
SOURCE: Annals of human genetics, (1983 Jul) 47 (Pt 3) 245-52.
Journal code: 0416661. ISSN: 0003-4800.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198310
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19831028

L4 ANSWER 19 OF 19 MEDLINE on STN
TI The modes of inheritance of insulin-dependent **diabetes** mellitus or the genetics of IDDM, no longer a nightmare but still a headache.
AB The discovery of HLA antigen associations with juvenile-type insulin-dependent **diabetes** mellitus (IDDM) provided strong evidence separating this disorder, or group of disorders, from maturity-type noninsulin-dependent **diabetes**, as well as adding to the evidence for an immunologic pathogenesis. In addition, it was hoped that the use of these **disease-marker** associations in appropriate studies might clarify the genetics of IDDM. While these associations have provided a useful tool to further investigate the genetics and pathogenesis of IDDM, the mode or modes of inheritance of this group of disorders remain an area of great controversy. Susceptibility to IDDM is currently being proposed as being inherited as a single autosomal dominant, as a single autosomal recessive, as recessive and some dominant forms, in an intermediate gene dosage model, in a heterogeneous three-allele or two HLA loci model, and as a two-locus disorder. The arguments for each of these proposals is presented, as well as the problems of each. We surmise that the weight of evidence supports the heterogeneity hypothesis but that the modes of inheritance of IDDM will be fully resolved only when we can more reliably identify the diabetogenic genotype, rather than being limited in our investigations to the study of only full-blown clinical disease.
ACCESSION NUMBER: 82110898 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7034532
TITLE: The modes of inheritance of insulin-dependent **diabetes** mellitus or the genetics of IDDM, no longer a nightmare but still a headache.
AUTHOR: Rotter J I

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CONTRACT NUMBER:    AM-00523 (NIADDK)
     AM-25834 (NIADDK)
SOURCE:             American journal of human genetics, (1981 Nov) 33 (6)
                    835-51.  Ref: 91
                    Journal code: 0370475. ISSN: 0002-9297.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    General Review; (REVIEW)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        198203
ENTRY DATE:         Entered STN: 19900317
                    Last Updated on STN: 19970203
                    Entered Medline: 19820313


=> d his

     (FILE 'HOME' ENTERED AT 13:31:35 ON 30 SEP 2004)

     FILE 'MEDLINE' ENTERED AT 13:31:41 ON 30 SEP 2004
L1             1 S OBESITY AND DISEASE MARKER
L2             0 S OSTEOPOROSIS AND DISEASE MARKER
L3             0 S L1 AND PROTEIN MARKER
L4            19 S DIABETES AND DISEASE MARKER
L5             0 S L4 AND PROTEIN MARKER

=> s HUSERFR3A
L6             0 HUSERFR3A

=> s (181 (S15)/HUSERFR3A)?
MISSING OPERATOR '181 (S15'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s diabetes and (non-genetic marker)
        206298 DIABETES
       3292412 NON
        502008 GENETIC
        119367 MARKER
             0 NON-GENETIC MARKER
                 (NON(W)GENETIC(W)MARKER)
L7             0 DIABETES AND (NON-GENETIC MARKER)

=> s diabetes and protein marker
        206298 DIABETES
       1311316 PROTEIN
        119367 MARKER
           225 PROTEIN MARKER
                 (PROTEIN(W)MARKER)
L8             3 DIABETES AND PROTEIN MARKER

=> d l8 ti abs ibib tot

L8   ANSWER 1 OF 3     MEDLINE on STN
TI   Retinopathy in type II diabetes mellitus associated with
     above-normal urinary excretion of RBP.
AB   We performed a cross-sectional study on the urinary excretion profiles of
     albumin (a marker of glomerular dysfunction) and retinol-binding protein
     (a low molecular mass protein marker of renal proximal
     tubular dysfunction) in non-insulin dependent (Type II) diabetics, with or
     without retinopathy.  The urinary excretion of both proteins, in
     particular retinol-binding protein, was significantly higher in patients
     with background/proliferative retinopathy compared to patients without
```

retinopathy. The degree of retinopathy correlated to the urinary excretion of albumin ($P < 0.005$) and retinol-binding protein ($P < 0.0001$). Retinopathy occurred at a higher frequency in patients with above-normal urinary excretion of retinol-binding protein, both in the absence or presence of micro/macroalbuminuria. The frequency of retinopathy among micro/macroalbuminuric patients with a normal urinary excretion of retinol-binding protein did not differ significantly from that observed in patients with a normal urinary excretion of both proteins. We cannot explain the association between retinopathy and proximal tubular dysfunction in Type II **diabetes**. However, it is possible that both phenomena are related to a common pathogenetic factor.

ACCESSION NUMBER: 95174269 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7869657
TITLE: Retinopathy in type II **diabetes** mellitus associated with above-normal urinary excretion of RBP.
AUTHOR: Holm J; Nielsen N V; Hemmingsen L
CORPORATE SOURCE: Department of Clinical Chemistry Central Hospital Nykobing Falster Nykobing, Denmark.
SOURCE: Kidney international. Supplement, (1994 Nov) 47 S105-8. Journal code: 7508622. ISSN: 0098-6577.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 19950407
Last Updated on STN: 19950407
Entered Medline: 19950329

L8 ANSWER 2 OF 3 MEDLINE on STN
TI Low-molecular-mass proteinuria as a marker of proximal renal tubular dysfunction in normo- and microalbuminuric non-insulin-dependent diabetic subjects.
AB We determined the urinary excretion, expressed as the protein/creatinine ratio (morning urines), of albumin (a marker of glomerular dysfunction) and retinol-binding protein (RBP; a low-molecular-mass **protein marker** of tubular proteinuria) in 102 non-insulin-dependent diabetic patients. There was a statistically significant ($P < 0.0001$) correlation (rho = 0.38) between the urinary excretion values of the two proteins. The population could be divided into four subgroups: 32 with normal excretion values, 15 with above-normal urinary excretion of RBP, 24 with above-normal urinary excretion of albumin, and 31 patients with above-normal urinary excretion of both proteins. No patients had above-normal serum creatinine concentrations or above-normal serum RBP concentrations. This seems to exclude "tubular overflow proteinuria" as the cause of the increased urinary excretion of RBP seen in some patients with non-insulin-dependent **diabetes**. Our data suggest the presence of a state of proximal tubular dysfunction in these patients.

ACCESSION NUMBER: 93193268 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8448868
TITLE: Low-molecular-mass proteinuria as a marker of proximal renal tubular dysfunction in normo- and microalbuminuric non-insulin-dependent diabetic subjects.
AUTHOR: Holm J; Hemmingsen L; Nielsen N V
CORPORATE SOURCE: Department of Clinical Chemistry, Central Hospital Nykobing Falster, Denmark.
SOURCE: Clinical chemistry, (1993 Mar) 39 (3) 517-9. Journal code: 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199304
ENTRY DATE: Entered STN: 19930423

Last Updated on STN: 19930423
Entered Medline: 19930415

L8 ANSWER 3 OF 3 MEDLINE on STN

TI Diabetic retinopathy related to degree of albuminuria and tubular (low molecular weight) proteinuria in insulin-dependent (type I) **diabetes** mellitus.

AB The urinary excretion of albumin (a marker of glomerular damage) and retinol binding protein (a low molecular weight **protein marker** of tubular dysfunction) was determined by sensitive immunochemical methods in 110 insulin-dependent (Type I) diabetic patients. We observed a statistically significant correlation between the urinary excretion levels of both proteins, in particular albumin, and the degree of retinopathy. The incidence of macroalbuminuria and tubular proteinuria was significantly higher in patients with manifest background retinopathy and proliferative retinopathy as compared to patients with no or slight retinopathy. The duration of **diabetes** was significantly correlated to the degree of retinopathy, but not to the urinary excretion of albumin and retinol binding protein.

ACCESSION NUMBER:	90364819 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 2392901
TITLE:	Diabetic retinopathy related to degree of albuminuria and tubular (low molecular weight) proteinuria in insulin-dependent (type I) **diabetes** mellitus.
AUTHOR:	Nielsen N V; Holm J; Hemmingsen L
CORPORATE SOURCE:	Department of Ophthalmology, Central Hospital Nykobing Falster, Denmark.
SOURCE:	Acta ophthalmologica, (1990 Jun) 68 (3) 270-4. Journal code: 0370347. ISSN: 0001-639X.
PUB. COUNTRY:	Denmark
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	199010
ENTRY DATE:	Entered STN: 19901109 Last Updated on STN: 19901109 Entered Medline: 19901004

=> file wpids, fsta, biosis, biotechds, hcaplus, dgene, embase, japio, jicst, fsta

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FULL ESTIMATED COST	7.34	7.55

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=> s diabetes and protein marker
   8 FILES SEARCHED...
L9   19 DIABETES AND PROTEIN MARKER

=> s obesity and protein marker
L10   3 OBESITY AND PROTEIN MARKER

=> s diabetes and (non-genetic marker)
L11   0 DIABETES AND (NON-GENETIC MARKER)

=> d 19 ti abs ibib tot

L9 ANSWER 1 OF 19 WPIDS COPYRIGHT 2004 THE THOMSON CORP on STN
TI New non-genetic based protein disease markers for obesity, osteoporosis, **diabetes**, osteoathritis and hypertension, useful in diagnosis and monitoring of treatment for these diseases and to screen for therapeutic compounds.
AN 2002-362307 [39] WPIDS
AB WO 200222165 A UPAB: 20020621
NOVELTY - Non-genetic based protein disease markers for obesity, osteoporosis, **diabetes**, osteoathritis and hypertension, are new.
DETAILED DESCRIPTION - Non-genetic based protein disease markers for obesity, osteoporosis, **diabetes**, osteoathritis and hypertension, are new, where markers for obesity (n=34), osteoporosis (n=20), **diabetes** (n=9), osteoathritis (n=1) and hypertension (n=9) are listed in the specification.
INDEPENDENT CLAIMS are also included for the following:
(1) determining a disease state of a subject suspected of having obesity, osteoporosis, **diabetes**, osteoathritis or hypertension comprising:
(a) obtaining a sample containing protein;
(b) measuring levels of protein markers of the disease state, where the markers are given in the specification; and
(c) comparing with levels in controls from disease-free subjects/control standards;
(2) binding reagents specific for the proteins, optionally bound to a detectable label;
(3) a standardized two-dimensional electrophoretic protein distribution from a sample (optionally human serum) from a subject having obesity, osteoporosis, **diabetes**, osteoathritis or hypertension (and optionally being treated with pharmaceuticals);
(4) protein markers comprising a composition of two or more proteins which individually do not have significantly different levels between disease/control samples in a method as in (1), but produce a combined value which is significantly different, and methods and binding reagents as in (1) and (2) relating to the markers;
(5) protein submarkers not altered statistically significantly in the method as in (1) but altered in tandem/opposite in level and direction to protein markers, and methods and binding reagents as in (1) and (2) relating to the markers;
(6) generating an index marker for a particular physiological state comprising:
(a) determining protein markers that differ between samples from a subject with a disease state and a control sample;
(b) selecting two or more of the markers;

(c) combining the values for the markers and determining where the combination of values is altered in a manner of greater statistical significance;

(7) index markers comprising two or more protein markers determined by (6);

(8) cloning a gene encoding a **protein marker** comprising:

(a) determining a partial amino acid sequence of the protein;

(b) deducing a nucleotide sequence for a gene encoding the protein; and

(c) isolating or synthesizing a gene encoding the nucleotide sequence; and

(9) polynucleotides encoding the proteins, and antisense sequences inhibiting gene expression.

ACTIVITY - Anorectic; osteopathic; antidiabetic; antiarthritic; hypotensive. No biological data is given.

MECHANISM OF ACTION - None given.

USE - The markers and a new method are useful to diagnose obesity, osteoporosis, **diabetes**, osteoathritis or hypertension in individuals. Marker levels may also be used to determine disease severity. The markers and method can also be used to monitor the efficacy of therapy for the conditions, by comparing marker levels between samples from a subject taken at different times. The markers identified may also be drug development targets for the diseases. The protein markers can be used to screen compounds for biological activity against the diseases, which may be included with a carrier in pharmaceutical compositions useful to treat the disease states. The markers are useful to screen candidate compounds for detection of or therapeutic activity against disease states, and to identify biological pathways involved in disease states. They are also useful to identify synergistic agents which may be included in pharmaceutical compositions (all claimed).

Dwg.0/10

ACCESSION NUMBER: 2002-362307 [39] WPIDS
DOC. NO. CPI: C2002-102544
TITLE: New non-genetic based protein disease markers for obesity, osteoporosis, **diabetes**, osteoathritis and hypertension, useful in diagnosis and monitoring of treatment for these diseases and to screen for therapeutic compounds.
DERWENT CLASS: B04 D16
INVENTOR(S): ANDERSON, N L; MYERS, T G; PIEPER, R; STEINER, S; TAYLOR, J; MYERS, T; REMBERT, P
PATENT ASSIGNEE(S): (ANDE-I) ANDERSON N L; (MYER-I) MYERS T G; (PIEP-I) PIEPER R; (STEI-I) STEINER S; (TAYL-I) TAYLOR J; (LARG-N) LARGE SCALE PROTEOMICS CORP
COUNTRY COUNT: 97
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002022165	A1	20020321	(200239)*	EN	63
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
US 2002072492	A1	20020613	(200243)		
AU 2001088973	A	20020326	(200251)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 2002022165	A1	WO 2001-US28268	20010912
US 2002072492	A1 CIP of	US 2000-660242	20000912
		US 2001-886271	20010622
AU 2001088973	A	AU 2001-88973	20010912

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001088973	A Based on	WO 2002022165

PRIORITY APPLN. INFO: US 2001-886271 20010622; US 2000-660242 20000912

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TI Serum C-reactive protein: A predictor of mortality in continuous ambulatory peritoneal dialysis (CAPD) patients.

ACCESSION NUMBER: 1998:23324 BIOSIS
DOCUMENT NUMBER: PREV199800023324
TITLE: Serum C-reactive protein: A predictor of mortality in continuous ambulatory peritoneal dialysis (CAPD) patients.
AUTHOR(S): Han, D. S.; Noh, H. J.; Shin, S. K.; Lee, I. H.; Kang, S. W.; Choi, K. H.; Lee, H. Y.
CORPORATE SOURCE: Dep. Internal Med., Inst. Kidney Disease, Yonsei Univ. Coll. Med., Seoul, South Korea
SOURCE: Journal of the American Society of Nephrology, (Sept., 1997) Vol. 9, No. PROGRAM AND ABSTR. ISSUE, pp. 264A. print.
Meeting Info.: 30th Annual Meeting of the American Society of Nephrology. San Antonio, Texas, USA. November 2-5, 1997. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Jan 1998
Last Updated on STN: 5 Jan 1998

L9 ANSWER 3 OF 19 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

TI Retinopathy in type II **diabetes** mellitus associated with above-normal urinary excretion of RBP.

AB We performed a cross-sectional study on the urinary excretion profiles of albumin (a marker of glomerular dysfunction) and retinol-binding protein (a low molecular mass **protein marker** of renal proximal tubular dysfunction) in non-insulin dependent (Type II) diabetics, with or without retinopathy. The urinary excretion of both proteins, in particular retinol-binding protein, was significantly higher in patients with background/proliferative retinopathy compared to patients without retinopathy. The degree of retinopathy correlated to the urinary excretion of albumin (P lt 0.005) and retinol-binding protein (P lt 0.0001). Retinopathy occurred at a higher frequency in patients with above-normal urinary excretion of retinol-binding protein, both in the absence or presence of micro/macroalbuminuria. The frequency of retinopathy among micro/macroalbuminuric patients with a normal urinary excretion of retinol-binding protein did not differ significantly from that observed in patients with a normal urinary excretion of both proteins. We cannot explain the association between retinopathy and proximal tubular dysfunction in Type II **diabetes**. However, it is possible that both phenomena are related to a common pathogenetic factor.

ACCESSION NUMBER: 1995:29042 BIOSIS

DOCUMENT NUMBER: PREV199598043342
TITLE: Retinopathy in type II **diabetes** mellitus associated with above-normal urinary excretion of RBP.
AUTHOR(S): Holm, Jan [Reprint author]; Nielsen, Niels Vesti; Hemmingsen, Lars
CORPORATE SOURCE: Dep. Clin. Chem., Cent. Hosp. Nykobing Falster, DK-4800 Nykobing Falster, Denmark
SOURCE: Kidney International Supplement, (1994) Vol. 0, No. 47, pp. S105-S108.
CODEN: KISUDF. ISSN: 0098-6577.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jan 1995
Last Updated on STN: 11 Jan 1995

L9 ANSWER 4 OF 19 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Low-molecular-mass proteinuria as a marker of proximal renal tubular dysfunction in normo- and microalbuminuric non-insulin-dependent diabetic subjects.
AB We determined the urinary excretion, expressed as the protein/creatinine ratio (morning urines), of albumin (a marker of glomerular dysfunction) and retinol-binding protein (RBP; a low-molecular-mass **protein marker** of tubular proteinuria) in 102 non-insulin-dependent diabetic patients. There was a statistically significant (P lt 0.0001) correlation (rho = 0.38) between the urinary excretion values of the two proteins. The population could be divided into four subgroups: 32 with normal excretion values, 15 with above-normal urinary excretion of RBP, 24 with above-normal urinary excretion of albumin, and 31 patients with above-normal serum creatinine concentrations or above-normal serum RBP concentrations. This seems to exclude "tubular overflow proteinuria" as the cause of the increased urinary excretion of RBP seen in some patients with non-insulin-dependent **diabetes**. Our data suggest the presence of a state of proximal tubular dysfunction in these patients.
ACCESSION NUMBER: 1993:281647 BIOSIS
DOCUMENT NUMBER: PREV199396011872
TITLE: Low-molecular-mass proteinuria as a marker of proximal renal tubular dysfunction in normo- and microalbuminuric non-insulin-dependent diabetic subjects.
AUTHOR(S): Holm, Jan [Reprint author]; Hemmingsen, Lars [Reprint author]; Nielsen, Niels V.
CORPORATE SOURCE: Dep. Clin. Chem., Cent. Hosp. Nykobing Falster, Nykobing Falster 4800, Denmark
SOURCE: Clinical Chemistry, (1993) Vol. 39, No. 3, pp. 517-519.
CODEN: CLCHAU. ISSN: 0009-9147.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Jun 1993
Last Updated on STN: 9 Jun 1993

L9 ANSWER 5 OF 19 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP. on STN
TI New biopolymer marker, useful for indicating, for determining risk-assessment or for identifying therapeutic avenues related to, a disease state e.g. Type II **diabetes**;
using drug screening, monoclonal antibody, peptide display and antibody library
AN 2003-19160 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - A biopolymer marker comprising a sequence (P1) having 11, 13 (each of the 3 sequences) or 20 amino acids or its analyte and which is useful in indicating at least one particular disease state, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) a method for evidencing and categorizing at least one disease state; (2) a diagnostic assay kit for determining the presence of the biopolymer

marker or for diagnosing, determining risk-assessment or identifying therapeutic avenues related to a disease state; (3) polyclonal antibodies produced against (P1) in at least one animal host; (4) a process for identifying therapeutic avenues related to a disease state; and (5) a process for regulating a disease state.

BIOTECHNOLOGY - Preferred Biopolymer Marker: The disease state indicated by the biopolymer marker is predictive of Type II **diabetes**. Preferred Antibody: The antibody is monoclonal or polyclonal antibody. Preferred Kit: The diagnostic assay kit for determining the presence of the biopolymer marker or for diagnosing, determining risk-assessment or identifying therapeutic avenues related to a disease state comprises: (a) at least one biochemical material that is capable of specifically binding with a biomolecule that includes the biopolymer marker related to the disease state; and (b) means for determining binding between the biochemical material and the biomolecule. At least one analysis to determine the presence of the biopolymer marker or a biochemical material specific for it is carried out on a sample. The biochemical material or molecule is immobilized on a solid support. It is labeled. It is a monoclonal antibody. Diagnosing, determining risk-assessment or identifying therapeutic avenues is carried out on a single sample or multiple samples, where the analysis is carried out on each of the first and second samples. The first and second samples are obtained at different time periods. Preferred Method: Evidencing and categorizing at least one disease state comprises: (a) obtaining a sample from a patient; (b) conducting mass spectrometric analysis on the sample; (c) evidencing and categorizing at least one biopolymer marker sequence or its analyte isolated from the sample; and (d) comparing the isolated biopolymer marker sequence or its analyte to (P1), where correlation of the isolated biopolymer marker and (P1) evidences and categorizes the at least one disease state. The step of evidencing and categorizing is particularly directed to biopolymer markers or analytes linked to at least one risk of disease development of the patient or to the existence of a particular disease state. The sample is an unfractionated body fluid or a tissue sample. It comprises blood, blood products, urine, saliva, cerebrospinal fluid or lymph. The mass spectrometric analysis is Surface Enhanced Laser Desorption Ionization (SELDI) mass spectrometry (MS), Maldi Qq TOF, MS/MS, TOF-TOF, ESI-Q-TOF or ION-TRAP. The patient is a human. Identifying therapeutic avenues related to a disease state comprises: (a) conducting an analysis as provided by the kit; and (b) interacting with the biopolymer. The therapeutic avenues include: (a) utilization and recognition of the biopolymer markers, or their variants or moieties as direct therapeutic modalities, either alone or in conjunction with a carrier; (b) validation of therapeutic modalities or disease preventive agents as a function of biopolymer marker presence or concentration; (c) treatment or prevention of a disease state by formation of disease intervention modalities; (d) use of biopolymer markers as a means of elucidating viable agents; (e) instigation of a therapeutic immunological response; or (f) synthesis of molecular structure related to the biopolymer markers, or their variants or moieties, which are constructed and arranged to intervene in the disease state. The treatment or prevention of a disease state by formation of disease intervention modalities is the formation of biopolymer/ligand conjugates, which intervene at receptor sites to prevent, delay or reverse a disease process. The means for elucidating viable agents includes use of a bacteriophage peptide display or antibody library. Regulating a disease state comprises controlling the presence or absence of the biopolymer or its analyte.

USE - The biopolymer marker is useful for indicating, for determining risk-assessment or for identifying therapeutic avenues related to, a disease state e.g., Type II **diabetes** (claimed).

EXAMPLE - No relevant examples given. (44 pages)

ACCESSION NUMBER: 2003-19160 BIOTECHDS

TITLE: New biopolymer marker, useful for indicating, for determining risk-assessment or for identifying therapeutic avenues

related to, a disease state e.g. Type II **diabetes**;
using drug screening, monoclonal antibody, peptide display and antibody library

AUTHOR: JACKOWSKI G; MARSHALL J
PATENT ASSIGNEE: SYN.X PHARMA INC
PATENT INFO: WO 2003045993 5 Jun 2003
APPLICATION INFO: WO 2002-CA1669 31 Oct 2002
PRIORITY INFO: US 2001-993393 23 Nov 2001; US 2001-993393 23 Nov 2001
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-513630 [48]

L9 ANSWER 6 OF 19 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP. on STN
TI Novel biopolymer marker useful in indicating at least one disease state particularly a disease recognized as Syndrome X related disease;
for use in disease diagnosis
AN 2003-11517 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - A biopolymer marker (I) useful in indicating at least one disease state, is new.

DETAILED DESCRIPTION - A biopolymer marker (I) useful in indicating at least one disease state, is new. (I) comprises a sequence (S1).
Asp-Ala-His-Lys-Ser-Glu-Val-Ala-His-Arg-Phe-Lys-Asp-Leu-Gly-Glu-Glu (S1)

BIOTECHNOLOGY - Preparation: (I) is isolated from serum using standard purification techniques.

USE - (I) is useful as biopolymer marker for indicating at least one disease state particularly a disease recognized as a Syndrome X related disease (claimed).

ADVANTAGE - Promulgation of various forms of risk assessment tests are contemplated using (I), to identify asymptomatic patients before they suffer an irreversible event such as **diabetes**, kidney failure and heart failure, and enable effective disease management and preventative medicine. Additionally, the specific diagnostic tests which evolve using (I) provide a tool for rapidly and accurately diagnosing acute Syndrome X such as heart attack and stroke, and facilitate treatment.

EXAMPLE - No suitable example given.(9 pages)

ACCESSION NUMBER: 2003-11517 BIOTECHDS
TITLE: Novel biopolymer marker useful in indicating at least one disease state particularly a disease recognized as Syndrome X related disease;
for use in disease diagnosis
AUTHOR: JACKOWSKI G; THATCHER B; MARSHALL J; YANTHA J; VREES T
PATENT ASSIGNEE: JACKOWSKI G; THATCHER B; MARSHALL J; YANTHA J; VREES T
PATENT INFO: US 2002160418 31 Oct 2002
APPLICATION INFO: US 2001-845727 30 Apr 2001
PRIORITY INFO: US 2001-845727 30 Apr 2001; US 2001-845727 30 Apr 2001
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-255192 [25]

L9 ANSWER 7 OF 19 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP. on STN
TI Novel secreted or transmembrane protein and polynucleotide encoding the protein, useful for diagnosis and treatment of neurological disorders, cancer, autoimmune diseases, bone disorders and lung or liver fibrosis;
vector-mediated gene transfer and expression in host cell for recombinant protein production and gene therapy
AN 2003-00702 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Secreted or transmembrane protein (I) of 68 fully defined proteins, especially 7 proteins (P1-P7) with sequence (S1) of 48, 359, 228, 51, 178, 268, or 462 amino acids and encoded by a cDNA insert of clones bd1647, bp7833, bf171-6, bl20910, en5398, ci254 and as2943 with ATCC 98364, 98369, 98371, 98379, 98408, 98415 and 98444, respectively, is

new.

DETAILED DESCRIPTION - (I) is chosen from 68 secreted or transmembrane proteins of specific amino acids given in the specification, their fragments, and is encoded by specific complementary deoxyribonucleic acid (cDNA) inserts, where the protein is substantially free from other mammalian proteins. INDEPENDENT CLAIMS are also included for: (1) an isolated polynucleotide (II) chosen from 61 polynucleotides, encoding the secreted or transmembrane protein, comprising specific nucleotides given in the specification, its fragment, allelic variant, species homolog or hybridizable sequence; (2) an isolated gene corresponding to cDNA sequence of (II); (3) an isolated polynucleotide (III) chosen from 7 polynucleotides comprising a sequence of 1800, 2199, 1521, 2355, 2754, 1480, and 1755 bp, respectively, their fragments, allelic variants, species homologs, hybridizable sequences, encoding proteins P1-P7 as above, or the full-length protein or mature protein encoded by the cDNA insert of clones bd1647, bp7833, bf171-6, bl20910, en5398, ci254 and as2943 deposited under accession number American type culture collection (ATCC) 98364, 98369, 98371, 98379, 98408, 98415 and 98444, respectively; (4) a host cell (IV) transformed with (III); (5) producing (M1) a protein encoded by (III); (6) a protein produced by (M1); and (7) a pharmaceutical composition comprising proteins, P1-P7.

BIOTECHNOLOGY - Preparation: P1-P7 is produced by culturing (IV) in a suitable culture medium and purifying the protein from the culture (claimed). Preferred Polynucleotide: (III) is operably linked to at least one expression control sequence. Preferred Protein: P1-P7 comprises a mature protein.

ACTIVITY - Cytostatic; Antirheumatic; Antiarthritic; Vulnerary; Antiinflammatory; Antibacterial; Immunosuppressive; Antiparkinsonian; Neuroprotective; Nootropic; Osteopathic; Hemostatic; Vasotropic; Antiulcer; Fungicide; Antidiabetic; Antiasthmatic; Antiallergic; Immunostimulant; Analgesic; Antiparasitic. No suitable data given.

MECHANISM OF ACTION - Gene therapy.

USE - Proteins P1-P4 (encoded by the cDNA inserts bd1647, bp7833, bf171-6, and bl20910) are useful for preventing, treating or ameliorating a medical condition (claimed). (I) is useful for the immunological treatment or prevention of tumors. (I) exhibits activity relating to angiogenesis, cytokine, cell proliferation, cell differentiation, antiinflammatory, stem cell growth factor activity and activin or inhibin-related activities. (I) can be used to manipulate stem cells in culture to give rise to neuroepithelial cells that can be used to augment or replace cells damaged by illness, autoimmune disease, accidental damage or genetic disorders. (I) induces the proliferation of neural cells and regeneration of nerve and brain tissue and is useful for the treatment of central and peripheral nervous system diseases and neuropathies, such as Alzheimer's, Parkinson's disease, Huntington's disease, amyotrophic lateral sclerosis. (I) is involved in chemotactic or chemokinetic activity, regulation of hematopoiesis and is useful for treating myeloid or lymphoid cell disorders, platelet disorders such as thrombocytopenia and for regeneration of bone, cartilage, tendon, ligament and/or nerve tissue growth, and in tissue repair, healing of burns, incisions, ulcers, for treating osteoporosis, osteoarthritis, bone degenerative disorders, or periodontal disease. (I) is also useful for gut protection or regeneration and treatment of lung or liver fibrosis, reperfusion injury in various tissues, various immune deficiencies and disorders including severe combined immunodeficiency (SCID), bacterial or fungal infections, autoimmune disorders e.g. multiple sclerosis, rheumatoid arthritis, **diabetes** mellitus, myasthenia gravis, allergic reactions and conditions, such as asthma or other respiratory problems. (I) exhibits activity relating to cytokine, cell proliferation, cell differentiation, immune stimulating or suppressing, hematopoiesis regulating, tissue growth, angiogenesis, activin or inhibin, chemotactic/chemokinetic, hemostatic, thrombolytic, receptor/ligand, antiinflammatory, tumor inhibition activities and other activities such as inhibiting the growth, infection, or function of or killing bacteria,

viruses, fungi, and other parasites, affecting bodily characteristics, including height, weight, hair color, eye color, skin, fat to lean ratio or other tissue pigmentation or organ or body part size or shape, effecting biorhythms or circardian cycles or rhythms, fertility of male or female subjects, metabolism, catabolism, anabolism, processing, utilization, storage or elimination of dietary fat, lipid, protein, carbohydrate, vitamins, and behavioral characteristics such as depression, analgesic and pain reducing effects, promoting differentiation and growth of embryonic stem cells in lineages other than hematopoietic lineages, hormonal or endocrine activity and immunoglobulin-like activity. (II) is useful to express recombinant protein, as markers for tissues in which the corresponding protein is preferentially expressed. As molecular weight markers on Southern gels, as chromosome markers or tags, as probes to hybridize and discover novel, related DNA sequences, as nutritional sources or supplements and as an antigen to raise anti-DNA antibodies or elicit another immune response.

ADMINISTRATION - Administered by oral, topical, subcutaneous, intraperitoneal, parenteral or intravenous injection at a dose of 0.01 mug-100 mg. (284 pages)

ACCESSION NUMBER: 2003-00702 BIOTECHDS

TITLE: Novel secreted or transmembrane protein and polynucleotide encoding the protein, useful for diagnosis and treatment of neurological disorders, cancer, autoimmune diseases, bone disorders and lung or liver fibrosis;
vector-mediated gene transfer and expression in host cell for recombinant protein production and gene therapy

AUTHOR: JACOBS K; MCCOY J M; LAVALLIE E R; COLLINS-RACIE L A; EVANS C; MERBERG D; TREACY M; SPAULDING V

PATENT ASSIGNEE: JACOBS K; MCCOY J M; LAVALLIE E R; COLLINS-RACIE L A; EVANS C; MERBERG D; TREACY M; SPAULDING V

PATENT INFO: US 2002065394 30 May 2002

APPLICATION INFO: US 2000-745763 22 Dec 2000

PRIORITY INFO: US 2000-745763 22 Dec 2000; US 1998-40963 18 Mar 1998

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2002-582343 [62]

L9 ANSWER 8 OF 19 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP. on STN

TI New non-genetic based protein disease markers for obesity, osteoporosis, **diabetes**, osteoathritis and hypertension, useful in diagnosis and monitoring of treatment for these diseases and to screen for therapeutic compounds;
two-dimensional electrophoresis and antisense oligonucleotide for protein distribution study, drug screening, proteomics analysis and potential gene therapy

AN 2002-12901 BIOTECHDS

AB DERWENT ABSTRACT:

NOVELTY - Non-genetic based protein disease markers for obesity, osteoporosis, **diabetes**, osteoathritis and hypertension, are new.

DETAILED DESCRIPTION - Non-genetic based protein disease markers for obesity, osteoporosis, **diabetes**, osteoathritis and hypertension, are new, where markers for obesity (n=34), osteoporosis (n=20), **diabetes** (n=9), osteoathritis (n=1) and hypertension (n=9) are listed in the specification. INDEPENDENT CLAIMS are also included for the following: (1) determining a disease state of a subject suspected of having obesity, osteoporosis, **diabetes**, osteoathritis or hypertension comprising: (a) obtaining a sample containing protein; (b) measuring levels of protein markers of the disease state, where the markers are given in the specification; and (c) comparing with levels in controls from disease-free subjects/control standards; (2) binding reagents specific for the proteins, optionally bound to a detectable label; (3) a standardized two-dimensional electrophoretic protein distribution from a sample (optionally human

serum) from a subject having obesity, osteoporosis, **diabetes**, osteoathritis or hypertension (and optionally being treated with pharmaceuticals); (4) protein markers comprising a composition of two or more proteins which individually do not have significantly different levels between disease/control samples in a method as in (1), but produce a combined value which is significantly different, and methods and binding reagents as in (1) and (2) relating to the markers; (5) protein submarkers not altered statistically significantly in the method as in (1) but altered in tandem/opposite in level and direction to protein markers, and methods and binding reagents as in (1) and (2) relating to the markers; (6) generating an index marker for a particular physiological state comprising: (a) determining protein markers that differ between samples from a subject with a disease state and a control sample; (b) selecting two or more of the markers; (c) combining the values for the markers and determining where the combination of values is altered in a manner of greater statistical significance; (7) index markers comprising two or more protein markers determined by (6); (8) cloning a gene encoding a **protein marker** comprising:
(a) determining a partial amino acid sequence of the protein; (b) deducing a nucleotide sequence for a gene encoding the protein; and (c) isolating or synthesizing a gene encoding the nucleotide sequence; and (9) polynucleotides encoding the proteins, and antisense sequences inhibiting gene expression.

BIOTECHNOLOGY - Preferred Proteins: The proteins are preferably isolated. Preparation: The protein markers may be detected by: (i) measuring levels of individual proteins in a proteome (i.e. a large number of proteins representing the total relevant portion and preferably all detectable proteins using a particular technique e.g. two-dimensional electrophoresis) of a sample; (ii) comparing with levels in the proteome of a control subject/control standard; and (iii) detecting if proteins are significantly (preferably p less than 0.001) increased/decreased. The proteins may be prepared by standard recombinant techniques. Preferred Methods: Methods of using the protein markers are described in the specification as follows: (a) to screen compounds for biological activity against obesity, osteoporosis, **diabetes**, osteoathritis or hypertension comprising contacting a candidate compound with a subject having one of the disease, measuring the level of the **protein marker**, and comparing the level of **protein marker** to the level of the marker in a control sample from a subject not having the disease state or a control standard; (b) to screen compounds for detection/therapeutic activity against a disease state comprising contacting a candidate compound with a **protein marker**, measuring the activity of the marker or the binding of the compound to the marker, and selecting for further development, compounds that affect activity or bind; (c) to identify biological pathways involved in a disease state comprising: (a) obtaining a biological sample from a subject having obesity, osteoporosis, **diabetes**, osteoathritis or hypertension; (b) determining levels of proteins in the proteome in the sample; (c) comparing the levels of each protein in the proteome to levels of protein in a control sample from a subject not having the disease state or a control standard; (d) determining which proteins have statistically higher or lower levels in each sample; (e) identifying several of the determined proteins; and (f) deducing which biological pathways are affected based on the identities of the proteins, where the biological pathways contain a protein having a statistically significant higher or lower level in a comparison between the 2 samples; and (d) to determine whether the effects of two agents are cumulative or synergistic comprising: (a) exposing a subject to a first agent and obtaining a protein containing biological sample; (b) exposing a subject to a second agent and obtaining a protein containing biological sample; (c) exposing a subject to a first and second agent and obtaining a biological sample; (d) measuring the levels of protein markers in each sample; (e) comparing the changes in levels of protein markers between a subject exposed to a first agent, a subject exposed to a second agent,

and a subject exposed to a first and second agent; and (f) determining whether the effects of the first agent and second agent are cumulative or synergistic.

ACTIVITY - Anorectic; osteopathic; antidiabetic; antiarthritic; hypotensive. No biological data is given.

MECHANISM OF ACTION - None given.

USE - The markers and a new method are useful to diagnose obesity, osteoporosis, **diabetes**, osteoathritis or hypertension in individuals. Marker levels may also be used to determine disease severity. The markers and method can also be used to monitor the efficacy of therapy for the conditions, by comparing marker levels between samples from a subject taken at different times. The markers identified may also be drug development targets for the diseases. The protein markers can be used to screen compounds for biological activity against the diseases, which may be included with a carrier in pharmaceutical compositions useful to treat the disease states. The markers are useful to screen candidate compounds for detection of or therapeutic activity against disease states, and to identify biological pathways involved in disease states. They are also useful to identify synergistic agents which may be included in pharmaceutical compositions (all claimed).

EXAMPLE - Four hundred pairs of monozygotic human twins were screened for phenotypic disease states, by measuring quantitive traits of: total fat mass and percent fat (obesity), insulin resistance (**diabetes**), spine and total bone mass density (osteoporosis), hip joint gap measurement (osteoarthritis), and central and radial blood pressure (hypertension). Seventy-nine twin pairs (158 subjects) were discordant for a disease state, and since twins were genetically identical the differences did not arise from a genetic process. Whole serum samples (25 micrograms for obesity and **diabetes** assessments, otherwise 50 microliters) having approximately 70 mg/ml proteins were subjected to proteometric analysis as described in the specification, in which the quantity of protein in a twin's sample was compared to its respective partner (if any) in the respective twin sample. Data were analyzed statistically by conventional methods for determining a correlation between each peturbed protein and disease state, and a list of significant markers for each respective disease state was generated, given in the specification. (63 pages)

ACCESSION NUMBER: 2002-12901 BIOTECHDS
TITLE: New non-genetic based protein disease markers for obesity, osteoporosis, **diabetes**, osteoathritis and hypertension, useful in diagnosis and monitoring of treatment for these diseases and to screen for therapeutic compounds; two-dimensional electrophoresis and antisense oligonucleotide for protein distribution study, drug screening, proteomics analysis and potential gene therapy
AUTHOR: REMBERT P; TAYLOR J; STEINER S; ANDERSON N L; MYERS T
PATENT ASSIGNEE: LARGE SCALE PROTEOMICS CORP
PATENT INFO: WO 2002022165 21 Mar 2002
APPLICATION INFO: WO 2000-US28268 12 Sep 2000
PRIORITY INFO: US 2001-886271 22 Jun 2001
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2002-362307 [39]

L9 ANSWER 9 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Should C-reactive protein be added to metabolic syndrome and to assessment of global cardiovascular risk?
AB A review. Of novel risk factors for cardiovascular disease currently under investigation, high-sensitivity C-reactive protein (hsCRP) is the most promising. To date, more than 20 prospective epidemiol. studies have demonstrated that hsCRP independently predicts vascular risk, 6 cohort studies have confirmed that hsCRP evaluation adds prognostic information beyond that available from the Framingham Risk Score, and 8 cohort studies have demonstrated additive prognostic value at all levels of metabolic

syndrome or in the prediction of type 2 **diabetes**. In contrast to several other biomarkers that also reflect biol. aspects of inflammation, hypofibrinolysis, and insulin resistance, hsCRP measurement is inexpensive, standardized, widely available, and has a decade-to-decade variation similar to that of cholesterol. Given the consistency of prognostic data for hsCRP and the practicality of its use in outpatient clin. settings, the authors believe the time has come for a careful consideration of adding hsCRP as a clin. criterion for metabolic syndrome and for the creation of an hsCRP-modified coronary risk score useful for global risk prediction in both men and women. Toward this end, the authors believe experts in the fields of epidemiol., prevention, vascular biol., and clin. cardiol. should be convened to begin discussing the merits of this proposal.

ACCESSION NUMBER: 2004:459690 HCAPLUS
DOCUMENT NUMBER: 141:188605
TITLE: Should C-reactive protein be added to metabolic syndrome and to assessment of global cardiovascular risk?
AUTHOR(S): Ridker, Paul M.; Wilson, Peter W. F.; Grundy, Scott M.
CORPORATE SOURCE: Donald W. Reynolds Center for Cardiovascular Research, Boston, MA, 02215, USA
SOURCE: Circulation (2004), 109(23), 2818-2825
CODEN: CIRCAZ; ISSN: 0009-7322
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 10 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Value of retinol binding protein in judgment of early renal damage in patients with **diabetes**
AB Objective: To explore the clin. significance of determining retinol binding protein (RBP) in diagnosis of early renal damage in patients with NIDDM. Methods: The urine levels of RBP, N-acetyl-β-D-glucosaminidase (NAG) and micro-albumin (mALB) were determined in patients with NIDDM. Results: The urine levels of RBP and NAG were significantly higher in patients with normal mALB and those with abnormal mALB than in controls ($P < 0.01$) . There was significant correlation among the 3 parameters. Conclusions: The sensitivity of RBP is higher than that of NAG and mALB. RBP is another sensitive index reflecting the renal tubular function. The detection of the 3 indexes in patients with NIDDM can distinguish the damages coming from renal tubules and renal globules to ensure early diagnosis of diabetic nephropathy.

ACCESSION NUMBER: 2003:844206 HCAPLUS
DOCUMENT NUMBER: 140:268765
TITLE: Value of retinol binding protein in judgment of early renal damage in patients with **diabetes**
AUTHOR(S): Zheng, Hongying; Zhu, Xinxing; Zhang, Xuezhi
CORPORATE SOURCE: Dep. of Lab. Tests, Central Hospital of Shengli Oil Field, Dongying, 257000, Peop. Rep. China
SOURCE: Zhongguo Yixue Jianyan Zazhi (2003), 4(4), 275-276
CODEN: ZYJZAL; ISSN: 1606-8025
PUBLISHER: Zhongguo Yixue Jianyan Zazhi Chuban Youxian Gongsi
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

L9 ANSWER 11 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI C-reactive protein predicts the deterioration of glycemia in Chinese subjects with impaired glucose tolerance
AB Recent studies have shown that C-reactive protein (CRP) predicts future risk of **diabetes** in healthy Caucasians. We determined whether plasma CRP level was elevated in Chinese subjects with impaired glucose tolerance (IGT) and whether CRP level could be used to predict progression to type 2

diabetes or reversion to normal glucose tolerance (NGT) in these high-risk individuals. A total of 228 subjects with IGT at baseline from the Hong Kong Cardiovascular Risk Factors Prevalence Study underwent repeat oral glucose tolerance testing after 2 yr. Plasma high-sensitivity CRP was measured from their stored baseline samples and from 228 subjects with NGT matched for age and BMI by an immunoturbidimetric assay. Subjects with IGT at baseline had higher plasma CRP levels than subjects with NGT: 1.18 mg/l (0.52-2.52) vs. 0.87 mg/l (0.37-1.84), median (interquartile range), P = 0.01. At 2 yr, 117 subjects with IGT reverted to NGT, 84 remained in IGT, and 21 progressed to **diabetes**. Individuals who progressed to **diabetes** had the highest plasma CRP levels at baseline (P < 0.0001). Those with baseline CRP levels in the third and top quartile had a relative risk of remaining in IGT or progressing to **diabetes** of 2.87 (95% CI 1.06-7.82) and 2.76 (1.06-7.31), resp., after adjusting for anthropometric measure and lifestyle factors. CRP independently predicts the risk of remaining in IGT or progressing to **diabetes** in Chinese subjects with IGT. CRP might provide an adjunctive measure for identifying subjects with the highest risk of progression to **diabetes** who would derive the greatest benefits from preventive interventions.

ACCESSION NUMBER: 2003:677776 HCAPLUS
DOCUMENT NUMBER: 139:290352

TITLE: C-reactive protein predicts the deterioration of glycemia in Chinese subjects with impaired glucose tolerance

AUTHOR(S): Tan, Kathryn C. B.; Wat, Nelson M. S.; Tam, Sidney C. F.; Janus, Edward D.; Lam, T. H.; Lam, Karen S. L.
CORPORATE SOURCE: Department of Medicine, Queen Mary Hospital, Hong Kong, Peop. Rep. China

SOURCE: Diabetes Care (2003), 26(8), 2323-2328
CODEN: DICAD2; ISSN: 0149-5992
PUBLISHER: American Diabetes Association, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 12 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Relevance of C-reactive protein levels in peritoneal dialysis patients
AB C-reactive protein (CRP) levels are increased in 30 to 50% of dialysis patients and predict cardiovascular morbidity and mortality. It is usually considered that raised CRP levels reflect underlying atherosclerosis. However, many patients may have clin. apparent cardiovascular disease without raised CRP levels. This study was designed to assess both the risk factors for high CRP levels and the usefulness of the test as a marker of clin. apparent coronary artery disease (CAD), peripheral vascular disease (PVD) and the presence of ongoing infections/inflammatory disorders (INF-INFL) in peritoneal dialysis patients. A chart review of 190 prevalent peritoneal dialysis patients was performed. CRP, albumin, ferritin, erythropoietin (EPO) dose and resistance, Kt/V, and residual renal function values were obtained and a history or presence of cardiovascular disease (CAD, PVD) and presence of INF-INFL recorded. Data were analyzed by Chi-square, Spearman correlation and logistic regression. A total of 31% of patients had a raised CRP. INF-INFL was highly predictive of raised CRP levels (OR 16.97; 95% CI 5.41 to 53.14, P = 0.000), whereas CAD and PVD either singly or in combination had no such association The sensitivity/specificity for CRP as a test for INF-INFL was 83/77%. For CAD and PVD, the sensitivities were less than 40% and specificities 70%. Increased CRP values were more common in females but not in diabetics. Weak linear correlations existed between CRP levels and albumin, ferritin and residual renal function (γ = -0.212, 0.228 and -0.163 resp., P < 0.02). By regression anal., INF-INFL predicted high CRP levels, but CAD and PVD did not. The majority of patients (57%) with high CRP had no identifiable cause; 40% of these

patients had subsequent or previous normal CRP values. High transport status predicted high CRP levels (OR 7.28; 95% CI 1.417 to 37.36, P = 0.006). The majority of elevated CRP levels in peritoneal dialysis patients occur without an obvious cause. Clin. apparent cardiovascular disease does not predict high CRP levels. CRP levels vary over time in the same patient, from normal to high or vice versa, for no obvious reason. Sources of inflammation other than CAD, PVD and clin. obvious INF-INFL in peritoneal dialysis patients remain to be identified.

ACCESSION NUMBER: 2002:131071 HCAPLUS
DOCUMENT NUMBER: 137:77228
TITLE: Relevance of C-reactive protein levels in peritoneal dialysis patients
AUTHOR(S): Fine, Adrian
CORPORATE SOURCE: Section of Nephrology, University of Manitoba, Winnipeg, MB, Can.
SOURCE: Kidney International (2002), 61(2), 615-620
CODEN: KDYIA5; ISSN: 0085-2538
PUBLISHER: Blackwell Publishing, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 13 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI β-Trace protein is not better than cystatin C as an indicator of reduced glomerular filtration rate
AB Cystatin C (CysC) and β-trace protein were compared as glomerular filtration rate (GFR) markers. Serum CysC from diabetic patients were analyzed by N latex Cystatin C, from Dade Behring, on the BNA analyzer, and Cystatin C PET KIt, from Dako. The ROC areas for CysC (Dade Behring) and β-trace protein did not differ significantly. The areas for CysC measured by Dako method and for creatinine were smaller compared with the Dade Behring method. Using samples stored at -80°, the Dade Behring cystatin C discriminates even better than β-trace protein. The results confirm that cystatin C may be the best low-mol. weight **protein marker** to indicate reduced GFR.

ACCESSION NUMBER: 2001:887525 HCAPLUS
DOCUMENT NUMBER: 136:148932
TITLE: β-Trace protein is not better than cystatin C as an indicator of reduced glomerular filtration rate
AUTHOR(S): Priem, Friedrich; Althaus, Harald; Jung, Klaus; Sinha, Pranav
CORPORATE SOURCE: Department of Laboratory Medicine, University Hospital Charite, Humboldt University Berlin, Berlin, 10098, Germany
SOURCE: Clinical Chemistry (Washington, DC, United States) (2001), 47(12), 2181
CODEN: CLCHAU; ISSN: 0009-9147
PUBLISHER: American Association for Clinical Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 14 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Low-molecular-mass proteinuria as a marker of proximal renal tubular dysfunction in normo- and microalbuminuric non-insulin-dependent diabetic subjects
AB The authors determined the urinary excretion, expressed as the protein/creatinine ratio (morning urines), of albumin (a marker of glomerular dysfunction) and retinol-binding protein (RBP; a low-mol.-mass **protein marker** of tubular proteinuria) in 102 non-insulin-dependent diabetic patients. There was a statistically significant correlation (ρ = 0.38) between the urinary excretion

values of the two proteins. The population could be divided into four subgroups: 32 with normal excretion values, 15 with above-normal urinary excretion of RBP, 24 with above-normal urinary excretion of albumin, and 31 patients with above-normal urinary excretion of both proteins. No patients had above-normal serum creatinine concns. or above-normal serum RBP concns. This seems to exclude "tubular overflow proteinuria" as the cause of the increased urinary excretion of RBP seen in some patients with non-insulin-dependent **diabetes**. The data suggest the presence of a state of proximal tubular dysfunction in these patients.

ACCESSION NUMBER: 1993:252517 HCAPLUS
DOCUMENT NUMBER: 118:252517
TITLE: Low-molecular-mass proteinuria as a marker of proximal renal tubular dysfunction in normo- and microalbuminuric non-insulin-dependent diabetic subjects
AUTHOR(S): Holm, Jan; Hemmingsen, Lars; Nielsen, Niels V.
CORPORATE SOURCE: Dep. Clin. Chem., Cent. Hosp. Nykoebing Falster, Nykoebing Falster, 4800, Den.
SOURCE: Clinical Chemistry (Washington, DC, United States) (1993), 39(3), 517-19
CODEN: CLCHAU; ISSN: 0009-9147
DOCUMENT TYPE: Journal
LANGUAGE: English

L9 ANSWER 15 OF 19 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Retinopathy in Type II **diabetes** mellitus associated with above-normal urinary excretion of RBP.

AB We performed a cross-sectional study on the urinary excretion profiles of albumin (a marker of glomerular dysfunction) and retinol-binding protein (a low molecular mass **protein marker** of renal proximal tubular dysfunction) in non-insulin dependent (Type II) diabetics, with or without retinopathy. The urinary excretion of both proteins, in particular retinol-binding protein, was significantly higher in patients with background/proliferative retinopathy compared to patients without retinopathy. The degree of retinopathy correlated to the urinary excretion of albumin ($P < 0.005$) and retinol-binding protein ($P < 0.0001$). Retinopathy occurred at a higher frequency in patients with above-normal urinary excretion of retinol-binding protein, both in the absence or presence of micro/macroalbuminuria. The frequency of retinopathy among micro/macroalbuminuric patients with a normal urinary excretion of retinol-binding protein did not differ significantly from that observed in patients with a normal urinary excretion of both proteins. We cannot explain the association between retinopathy and proximal tubular dysfunction in Type II **diabetes**. However, it is possible that both phenomena are related to a common pathogenetic factor.

ACCESSION NUMBER: 94333684 EMBASE
DOCUMENT NUMBER: 1994333684
TITLE: Retinopathy in Type II **diabetes** mellitus associated with above-normal urinary excretion of RBP.
AUTHOR: Holm J.; Nielsen N.V.; Hemmingsen L.
CORPORATE SOURCE: Department of Clinical Chemistry, Central Hospital,DK-4800 Nykobing Falster, Denmark
SOURCE: Kidney International, Supplement, (1994) -/47 (S-105-S-108).
ISSN: 0098-6577 CODEN: KISUDF
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 006 Internal Medicine
012 Ophthalmology
028 Urology and Nephrology
LANGUAGE: English
SUMMARY LANGUAGE: English

L9 ANSWER 16 OF 19 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Ability of neutrophils to produce active oxygen species in diabetic patients.

AB The stimulated productions of superoxide anion in polymorphonuclear leukocytes obtained from diabetic patients and control subjects were measured. Superoxide production was estimated with a flowcytometer by measuring oxidized fluorescent products of 2,7-dichlorofluorescin diacetate incorporated in leukocytes. The superoxide production stimulated by phorbol myristate acetate (PMA) was significantly less in diabetic patient's leukocytes than in control subjects' leukocytes (mean ± SE 12900 ± 257 versus 11400 ± 463 counts; $p<0.01$), and that stimulated by opsonized zymozan also tended to be less in diabetic patients' leukocytes than in those of control subjects. The PMA stimulated polymorphonuclear leukocytes was negatively correlated with fasted blood glucose levels in the diabetic patients ($r = -0.500$, $p<0.05$), but not with glycated **protein marker** levels. Stimulated superoxide production in polymorphonuclear leukocytes decreased after incubation with glucose at concentrations between 20 to 50mmol/l ($p<0.05$). We conclude that stimulated superoxide anion production in polymorphonuclear leukocytes was impaired by hyperglycemia. This impairment may be the consequence of a shrunken NADPH pool depleted via the sorbitol pathway in conditions of hyperglycemia and thought to be one of the reasons for the vulnerability to infection of diabetic patients.

ACCESSION NUMBER: 93349502 EMBASE
DOCUMENT NUMBER: 1993349502
TITLE: Ability of neutrophils to produce active oxygen species in diabetic patients.
AUTHOR: Kanno K.; Tokunaga K.; Ochi M.; Shishino K.; Murase M.; Saheki S.; Takeuchi N.; Shinohara R.; Ishiguro I.
CORPORATE SOURCE: Department of Clinical Laboratory, Ehime University Hospital,Ehime, Japan
SOURCE: Japanese Journal of Clinical Chemistry, (1993) 22/3 (168-172).
ISSN: 0370-5633 CODEN: RIKAAN
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
025 Hematology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English; Japanese

L9 ANSWER 17 OF 19 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Low-molecular-mass proteinuria as a marker of proximal renal tubular dysfunction in normo- and microalbuminuric non-insulin-dependent diabetic subjects.

AB We determined the urinary excretion, expressed as the protein/creatinine ratio (morning urines), of albumin (a marker of glomerular dysfunction) and retinol-binding protein (RBP; a low-molecular-mass **protein marker** of tubular proteinuria) in 102 non-insulin-dependent diabetic patients. There was a statistically significant ($P<0.0001$) correlation ($\rho = 0.38$) between the urinary excretion values of the two proteins. The population could be divided into four subgroups: 32 with normal excretion values, 15 with above-normal urinary excretion of RBP, 24 with above-normal urinary excretion of albumin, and 31 patients with above-normal urinary excretion of both proteins. No patients had above-normal serum creatinine concentrations or above-normal serum RBP concentrations. This seems to exclude 'tubular overflow proteinuria' as the cause of the increased urinary excretion of RBP seen in some patients with non-insulin-dependent **diabetes**. Our data suggest the presence of a state of proximal tubular dysfunction in these patients.

ACCESSION NUMBER: 93099023 EMBASE

DOCUMENT NUMBER: 1993099023
TITLE: Low-molecular-mass proteinuria as a marker of proximal renal tubular dysfunction in normo- and microalbuminuric non-insulin-dependent diabetic subjects.
AUTHOR: Holm J.; Hemmingsen L.; Nielsen N.V.
CORPORATE SOURCE: Department of Clinical Chemistry, Central Hospital Nykobing Falster,Nykobing Falster 4800, Denmark
SOURCE: Clinical Chemistry, (1993) 39/3 (517-519).
ISSN: 0009-9147 CODEN: CLCHAU
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
028 Urology and Nephrology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L9 ANSWER 18 OF 19 EMBASE
on STN
TI Diabetic retinopathy related to degree of albuminuria and tubular (low molecular weight) proteinuria in insulin-dependent (Type I) **diabetes** mellitus.
AB The urinary excretion of albumin (a marker of glomerular damage) and retinol binding protein (a low molecular weight **protein** **marker** of tubular dysfunction) was determined by sensitive immunochemical methods in 110 insulin-dependent (Type I) diabetic patients. We observed a statistically significant correlation between the urinary excretion levels of both proteins, in particular albumin, and the degree of retinopathy. The incidence of macroalbuminuria and tubular proteinuria was significantly higher in patients with manifest background retinopathy and proliferative retinopathy as compared to patients with no or slight retinopathy. The duration of **diabetes** was significantly correlated to the degree of retinopathy, but not to the urinary excretion of albumin and retinol binding protein.
ACCESSION NUMBER: 90216010 EMBASE
DOCUMENT NUMBER: 1990216010
TITLE: Diabetic retinopathy related to degree of albuminuria and tubular (low molecular weight) proteinuria in insulin-dependent (Type I) **diabetes** mellitus.
AUTHOR: Nielsen N.V.; Holm J.; Hemmingsen L.
CORPORATE SOURCE: Department of Ophthalmology, Central Hospital Nykobing Falster, Nykobing Falster, Denmark
SOURCE: Acta Ophthalmologica, (1990) 68/3 (270-274).
ISSN: 0001-639X CODEN: ACOPAT
COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
012 Ophthalmology
LANGUAGE: English
SUMMARY LANGUAGE: English

L9 ANSWER 19 OF 19 JICST-EPlus on STN
TI Ability of Neutrophils to Produce Active Oxygen Species in Diabetic Patients.
AB The stimulated productions of superoxide anion in polymorphonuclear leukocytes obtained from diabetic patients and control subjects were measured. Superoxide production was estimated with a flowcytometer by measuring oxidized fluorescent products of 2,7-dichlorofluorescin diacetate incorporated in leukocytes. The superoxide production stimulated by phorbol myristate acetate(PMA) was significantly less in diabetic patient's leukocytes than in control subjects' leukocytes (mean±SE 12900±257 versus 11400±463 counts; $p<0.01$), and that stimulated by opsonized zymozan also tended to be less in diabetic patients' leukocytes than in those of control subjects. The PMA stimulated polymorphonuclear

leukocytes was negatively correlated with fasted blood glucose levels in the diabetic patients (r=-0.500, p<0.05), but not with glycated **protein marker** levels. Stimulated superoxide production in polymorphonuclear leukocytes decreased after incubation with glucose at concentrations between 20 to 50mmol/l (p<0.05). We conclude that stimulated superoxide anion production in polymorphonuclear leukocytes was impaired by hyperglycemia. This impairment may be the consequence of a shrunken NADPH pool depleted via the sorbitol pathway in conditions of hyperglycemia and thought to be one of the reasons for the vulnerability to infection of diabetic patients. (author abst.)

ACCESSION NUMBER: 930914055 JICST-EPlus
TITLE: Ability of Neutrophils to Produce Active Oxygen Species in Diabetic Patients.
AUTHOR: KANNO KAZUHISA; TOKUNAGA KENJI; OCHI MASAAKI; SHISHINO KOJI; MURASE MITSUHARU; SAEKI SHUICHI; TAKEUCHI NOZOMU
SHINOHARA RIKIO
ISHIGURO ISAO
CORPORATE SOURCE: Ehime Univ., School of Medicine, Hospital
Fujitahoken'eiseidai Eisei
Fujitahoken'eiseidai I
SOURCE: Rinsho Kagaku (Japanese Journal of Clinical Chemistry), (1993) vol. 22, no. 3, pp. 168-172. Journal Code: Z0312B (Fig. 3, Tbl. 1, Ref. 12)
CODEN: RIKAAN; ISSN: 0370-5633
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

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L10 ANSWER 1 OF 3 WPIDS COPYRIGHT 2004 THE THOMSON CORP on STN
TI New non-genetic based protein disease markers for **obesity**, osteoporosis, diabetes, osteoathritis and hypertension, useful in diagnosis and monitoring of treatment for these diseases and to screen for therapeutic compounds.
AN 2002-362307 [39] WPIDS
AB WO 200222165 A UPAB: 20020621
NOVELTY - Non-genetic based protein disease markers for **obesity**, osteoporosis, diabetes, osteoathritis and hypertension, are new.

DETAILED DESCRIPTION - Non-genetic based protein disease markers for **obesity**, osteoporosis, diabetes, osteoathritis and hypertension, are new, where markers for **obesity** (n=34), osteoporosis (n=20), diabetes (n=9), osteoathritis (n=1) and hypertension (n=9) are listed in the specification.

INDEPENDENT CLAIMS are also included for the following:

(1) determining a disease state of a subject suspected of having **obesity**, osteoporosis, diabetes, osteoathritis or hypertension comprising:

(a) obtaining a sample containing protein;

(b) measuring levels of protein markers of the disease state, where the markers are given in the specification; and

(c) comparing with levels in controls from disease-free subjects/control standards;

(2) binding reagents specific for the proteins, optionally bound to a detectable label;

(3) a standardized two-dimensional electrophoretic protein distribution from a sample (optionally human serum) from a subject having **obesity**, osteoporosis, diabetes, osteoathritis or hypertension (and optionally being treated with pharmaceuticals);

(4) protein markers comprising a composition of two or more proteins which individually do not have significantly different levels between disease/control samples in a method as in (1), but produce a combined

value which is significantly different, and methods and binding reagents as in (1) and (2) relating to the markers;

(5) protein submarkers not altered statistically significantly in the method as in (1) but altered in tandem/opposite in level and direction to protein markers, and methods and binding reagents as in (1) and (2) relating to the markers;

(6) generating an index marker for a particular physiological state comprising:

(a) determining protein markers that differ between samples from a subject with a disease state and a control sample;

(b) selecting two or more of the markers;

(c) combining the values for the markers and determining where the combination of values is altered in a manner of greater statistical significance;

(7) index markers comprising two or more protein markers determined by (6);

(8) cloning a gene encoding a **protein marker** comprising:

(a) determining a partial amino acid sequence of the protein;

(b) deducing a nucleotide sequence for a gene encoding the protein; and

(c) isolating or synthesizing a gene encoding the nucleotide sequence; and

(9) polynucleotides encoding the proteins, and antisense sequences inhibiting gene expression.

ACTIVITY - Anorectic; osteopathic; antidiabetic; antiarthritic; hypotensive. No biological data is given.

MECHANISM OF ACTION - None given.

USE - The markers and a new method are useful to diagnose **obesity**, osteoporosis, diabetes, osteoathritis or hypertension in individuals. Marker levels may also be used to determine disease severity. The markers and method can also be used to monitor the efficacy of therapy for the conditions, by comparing marker levels between samples from a subject taken at different times. The markers identified may also be drug development targets for the diseases. The protein markers can be used to screen compounds for biological activity against the diseases, which may be included with a carrier in pharmaceutical compositions useful to treat the disease states. The markers are useful to screen candidate compounds for detection of or therapeutic activity against disease states, and to identify biological pathways involved in disease states. They are also useful to identify synergistic agents which may be included in pharmaceutical compositions (all claimed).

Dwg.0/10

ACCESSION NUMBER: 2002-362307 [39] WPIDS
DOC. NO. CPI: C2002-102544
TITLE: New non-genetic based protein disease markers for **obesity**, osteoporosis, diabetes, osteoathritis and hypertension, useful in diagnosis and monitoring of treatment for these diseases and to screen for therapeutic compounds.
DERWENT CLASS: B04 D16
INVENTOR(S): ANDERSON, N L; MYERS, T G; PIEPER, R; STEINER, S; TAYLOR, J; MYERS, T; REMBERT, P
PATENT ASSIGNEE(S): (ANDE-I) ANDERSON N L; (MYER-I) MYERS T G; (PIEP-I) PIEPER R; (STEI-I) STEINER S; (TAYL-I) TAYLOR J; (LARG-N) LARGE SCALE PROTEOMICS CORP
COUNTRY COUNT: 97
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002022165	A1	20020321	(200239)*	EN	63

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO
RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

US 2002072492 A1 20020613 (200243)
AU 2001088973 A 20020326 (200251)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002022165	A1	WO 2001-US28268	20010912
US 2002072492	A1 CIP of	US 2000-660242	20000912
		US 2001-886271	20010622
AU 2001088973	A	AU 2001-88973	20010912

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001088973	A	Based on	WO 2002022165

PRIORITY APPLN. INFO: US 2001-886271 20010622; US 2000-660242 20000912

L10 ANSWER 2 OF 3 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP. on STN

TI New non-genetic based protein disease markers for **obesity**, osteoporosis, diabetes, osteoathritis and hypertension, useful in diagnosis and monitoring of treatment for these diseases and to screen for therapeutic compounds;
two-dimensional electrophoresis and antisense oligonucleotide for protein distribution study, drug screening, proteomics analysis and potential gene therapy

AN 2002-12901 BIOTECHDS

AB DERWENT ABSTRACT:

NOVELTY - Non-genetic based protein disease markers for **obesity**, osteoporosis, diabetes, osteoathritis and hypertension, are new.

DETAILED DESCRIPTION - Non-genetic based protein disease markers for **obesity**, osteoporosis, diabetes, osteoathritis and hypertension, are new, where markers for **obesity** (n=34), osteoporosis (n=20), diabetes (n=9), osteoathritis (n=1) and hypertension (n=9) are listed in the specification. INDEPENDENT CLAIMS are also included for the following: (1) determining a disease state of a subject suspected of having **obesity**, osteoporosis, diabetes, osteoathritis or hypertension comprising: (a) obtaining a sample containing protein; (b) measuring levels of protein markers of the disease state, where the markers are given in the specification; and (c) comparing with levels in controls from disease-free subjects/control standards; (2) binding reagents specific for the proteins, optionally bound to a detectable label; (3) a standardized two-dimensional electrophoretic protein distribution from a sample (optionally human serum) from a subject having **obesity**, osteoporosis, diabetes, osteoathritis or hypertension (and optionally being treated with pharmaceuticals); (4) protein markers comprising a composition of two or more proteins which individually do not have significantly different levels between disease/control samples in a method as in (1), but produce a combined value which is significantly different, and methods and binding reagents as in (1) and (2) relating to the markers; (5) protein submarkers not altered statistically significantly in the method as in (1) but altered in tandem/opposite in level and direction to protein markers, and methods and binding reagents as in (1) and (2) relating to the markers; (6) generating an index marker for a particular physiological state comprising: (a) determining protein markers that differ between samples from a subject with a disease state and a control sample; (b) selecting

two or more of the markers; (c) combining the values for the markers and determining where the combination of values is altered in a manner of greater statistical significance; (7) index markers comprising two or more protein markers determined by (6); (8) cloning a gene encoding a **protein marker** comprising: (a) determining a partial amino acid sequence of the protein; (b) deducing a nucleotide sequence for a gene encoding the protein; and (c) isolating or synthesizing a gene encoding the nucleotide sequence; and (9) polynucleotides encoding the proteins, and antisense sequences inhibiting gene expression.

BIOTECHNOLOGY - Preferred Proteins: The proteins are preferably isolated. Preparation: The protein markers may be detected by: (i) measuring levels of individual proteins in a proteome (i.e. a large number of proteins representing the total relevant portion and preferably all detectable proteins using a particular technique e.g. two-dimensional electrophoresis) of a sample; (ii) comparing with levels in the proteome of a control subject/control standard; and (iii) detecting if proteins are significantly (preferably p less than 0.001) increased/decreased. The proteins may be prepared by standard recombinant techniques. Preferred Methods: Methods of using the protein markers are described in the specification as follows: (a) to screen compounds for biological activity against **obesity**, osteoporosis, diabetes, osteoathritis or hypertension comprising contacting a candidate compound with a subject having one of the disease, measuring the level of the **protein marker**, and comparing the level of **protein marker** to the level of the marker in a control sample from a subject not having the disease state or a control standard; (b) to screen compounds for detection/therapeutic activity against a disease state comprising contacting a candidate compound with a **protein marker**, measuring the activity of the marker or the binding of the compound to the marker, and selecting for further development, compounds that affect activity or bind; (c) to identify biological pathways involved in a disease state comprising: (a) obtaining a biological sample from a subject having **obesity**, osteoporosis, diabetes, osteoathritis or hypertension; (b) determining levels of proteins in the proteome in the sample; (c) comparing the levels of each protein in the proteome to levels of protein in a control sample from a subject not having the disease state or a control standard; (d) determining which proteins have statistically higher or lower levels in each sample; (e) identifying several of the determined proteins; and (f) deducing which biological pathways are affected based on the identities of the proteins, where the biological pathways contain a protein having a statistically significant higher or lower level in a comparison between the 2 samples; and (d) to determine whether the effects of two agents are cumulative or synergistic comprising: (a) exposing a subject to a first agent and obtaining a protein containing biological sample; (b) exposing a subject to a second agent and obtaining a protein containing biological sample; (c) exposing a subject to a first and second agent and obtaining a biological sample; (d) measuring the levels of protein markers in each sample; (e) comparing the changes in levels of protein markers between a subject exposed to a first agent, a subject exposed to a second agent, and a subject exposed to a first and second agent; and (f) determining whether the effects of the first agent and second agent are cumulative or synergistic.

ACTIVITY - Anorectic; osteopathic; antidiabetic; antiarthritic; hypotensive. No biological data is given.

MECHANISM OF ACTION - None given.

USE - The markers and a new method are useful to diagnose **obesity**, osteoporosis, diabetes, osteoathritis or hypertension in individuals. Marker levels may also be used to determine disease severity. The markers and method can also be used to monitor the efficacy of therapy for the conditions, by comparing marker levels between samples from a subject taken at different times. The markers identified may also be drug development targets for the diseases. The protein markers can be used to screen compounds for biological activity against the diseases,

which may be included with a carrier in pharmaceutical compositions useful to treat the disease states. The markers are useful to screen candidate compounds for detection of or therapeutic activity against disease states, and to identify biological pathways involved in disease states. They are also useful to identify synergistic agents which may be included in pharmaceutical compositions (all claimed).

EXAMPLE - Four hundred pairs of monozygotic human twins were screened for phenotypic disease states, by measuring quantitive traits of: total fat mass and percent fat (**obesity**), insulin resistance (diabetes), spine and total bone mass density (osteoporosis), hip joint gap measurement (osteoarthritis), and central and radial blood pressure (hypertension). Seventy-nine twin pairs (158 subjects) were discordant for a disease state, and since twins were genetically identical the differences did not arise from a genetic process. Whole serum samples (25 micrograms for **obesity** and diabetes assessments, otherwise 50 microliters) having approximately 70 mg/ml proteins were subjected to proteometric analysis as described in the specification, in which the quantity of protein in a twin's sample was compared to its respective partner (if any) in the respective twin sample. Data were analyzed statistically by conventional methods for determining a correlation between each peturbed protein and disease state, and a list of significant markers for each respective disease state was generated, given in the specification. (63 pages)

ACCESSION NUMBER: 2002-12901 BIOTECHDS
TITLE: New non-genetic based protein disease markers for **obesity**, osteoporosis, diabetes, osteoathritis and hypertension, useful in diagnosis and monitoring of treatment for these diseases and to screen for therapeutic compounds; two-dimensional electrophoresis and antisense oligonucleotide for protein distribution study, drug screening, proteomics analysis and potential gene therapy
AUTHOR: REMBERT P; TAYLOR J; STEINER S; ANDERSON N L; MYERS T
PATENT ASSIGNEE: LARGE SCALE PROTEOMICS CORP
PATENT INFO: WO 2002022165 21 Mar 2002
APPLICATION INFO: WO 2000-US28268 12 Sep 2000
PRIORITY INFO: US 2001-886271 22 Jun 2001
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2002-362307 [39]

L10 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Association of C-reactive protein with markers of prevalent atherosclerotic disease
AB Recent prospective studies have demonstrated that elevated C-reactive protein (CRP) is a marker of increased risk of atherothrombotic clin. events. We examined in a large, cross-sectional family-based study (n = 875 men, 948 women) whether serum CRP was associated with prevalent coronary heart disease (CHD), the ankle/brachial blood pressure index, or carotid intima-media thickness, an indicator of subclin. atherosclerosis as assessed by B-mode ultrasound. CRP was associated with many other cardiovascular risk factors, particularly markers of **obesity** and insulin resistance, markers of inflammation and acute phase reaction, and hormone replacement therapy. Adjusted for age and family type, there was a weak pos. association of CRP with carotid intima-media thickness in both genders and with prevalent CHD in women. However, adjustment for other risk factors completely eliminated the assocns. For example, among women, the risk factor-adjusted mean values of intima-media thickness across quartiles of CRP were 0.76, 0.74, 0.75, and 0.76 mm ($p > 0.5$). In men there was a weak inverse association between CRP and ankle/brachial blood pressure index, independent of other risk factors, but no such association in women. Our findings indicate that CRP is not strongly and independently associated with prevalent atherosclerosis. Because CRP has been associated with clin. events, it could be that elevated CRP may be a stronger marker of

thrombotic risk than of the degree of atherosclerosis.

ACCESSION NUMBER: 2001:503287 HCAPLUS
DOCUMENT NUMBER: 136:165083
TITLE: Association of C-reactive protein with markers of prevalent atherosclerotic disease
AUTHOR(S): Folsom, A. R.; Pankow, J. S.; Tracy, R. P.; Arnett, D. K.; Peacock, J. M.; Hong, Y.; Djousse, L.; Eckfeldt, J. H.
CORPORATE SOURCE: Investigators of the NHLBI Family Heart Study, Division of Epidemiology, School of Public Health, University of Minnesota, Minneapolis, MN, USA
SOURCE: American Journal of Cardiology (2001), 88(2), 112-117
CODEN: AJCDAG; ISSN: 0002-9149
PUBLISHER: Excerpta Medica, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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=> e myers
E1            1     MYERPEROXIDASE/BI
E2            1     MYERROR/BI
E3        11251 --> MYERS/BI
E4            1     MYERSAE/BI
E5            2     MYERSALNA/BI
E6           10     MYERSCOUGH/BI
E7            2     MYERSGLANIS/BI
E8          131     MYERSI/BI
E9            1     MYERSIA/BI
E10          11     MYERSIANA/BI
E11           5     MYERSIELLA/BI
E12          11     MYERSII/BI

=> s e3
L12       11251 MYERS/BI

=> s l12 and l1
L13           0 L12 AND L1

=> s disease marker adj protein
L14           0 DISEASE MARKER ADJ PROTEIN
```

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 12578
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 7 USPATFULL on STN
TI Schizophrenia associated gene, proteins and biallelic markers
AB The invention concerns the human g35030 gene, polynucleotides, polypeptides biallelic markers, and human chromosome 13q31-q33 biallelic markers. The invention also concerns the association established between schizophrenia and bipolar disorder and the biallelic markers and the g35030 gene and nucleotide sequences. The invention provides means to identify compounds useful in the treatment of schizophrenia, bipolar disorder and related diseases, means to determine the predisposition of individuals to said disease as well as means for the disease diagnosis and prognosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:115714 USPATFULL
TITLE: Schizophrenia associated gene, proteins and biallelic markers
INVENTOR(S): Cohen, Daniel, Neuilly-sur-Seine, FRANCE
Blumenfeld, Marta, Paris, FRANCE
Chumakov, Ilya, Vaux-le-Penil, FRANCE
Bougueleret, Lydie, Petit Lancy, SWITZERLAND
Essioux, Laurent, Paris, FRANCE
PATENT ASSIGNEE(S): Genset S.A., FRANCE (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6555316	B1	20030429
APPLICATION INFO.:	US 2000-679409		20001003 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-539333, filed on 30 Mar 2000, now patented, Pat. No. US 6476208
Continuation-in-part of Ser. No. US 1999-416384, filed on 12 Oct 1999

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-168088P	19991130 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Fredman, Jeffrey
LEGAL REPRESENTATIVE: Saliwanchik, Lloyd & Saliwanchik
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Figure(s); 15 Drawing Page(s)
LINE COUNT: 9055
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 4 OF 7 USPATFULL on STN
TI Genomic sequence of the 5-lipoxygenase-activating protein (FLAP), polymorphic markers thereof and methods for detection of asthma
AB The invention concerns the genomic sequence of the FLAP gene. The invention also concerns biallelic markers of a FLAP gene and the association established between these markers and diseases involving the leukotriene pathway such as asthma. The invention provides means to determine the predisposition of individuals to diseases involving the leukotriene pathway as well as means for the diagnosis of such diseases and for the prognosis/detection of an eventual treatment response to agents acting on the leukotriene pathway.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:67651 USPATFULL

TITLE: Genomic sequence of the 5-lipoxygenase-activating protein (FLAP), polymorphic markers thereof and methods for detection of asthma
INVENTOR(S): Blumenfeld, Marta, Paris, FRANCE
Chumakov, Ilya, Vaux-le-Penil, FRANCE
Bougueleret, Lydie, Vanves, FRANCE
PATENT ASSIGNEE(S): Genset S.A., FRANCE (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6531279	B1	20030311
APPLICATION INFO.:	US 1999-292542		19990415 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-81893P	19980415 (60)
	US 1998-91314P	19980630 (60)
	US 1999-123406P	19990308 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Goldberg, Jeanine
LEGAL REPRESENTATIVE: Saliwanchik, Lloyd & Saliwanchik
NUMBER OF CLAIMS: 3
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 7280
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 5 OF 7 USPATFULL on STN
TI Schizophrenia associated genes, proteins and biallelic markers
AB The invention concerns the human sbg1, g34665, sbg2, g35017 and g35018 genes, polynucleotides, polypeptides biallelic markers, and human chromosome 13q31-q33 biallelic markers. The invention also concerns the association established between schizophrenia and bipolar disorder and the biallelic markers and the sbg1, g34665, sbg2, g35017 and g35018 genes and nucleotide sequences. The invention provides means to identify compounds useful in the treatment of schizophrenia, bipolar disorder and related diseases, means to determine the predisposition of individuals to said disease as well as means for the disease diagnosis and prognosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:291075 USPATFULL
TITLE: Schizophrenia associated genes, proteins and biallelic markers
INVENTOR(S): Cohen, Daniel, Neuilly-Sue-Seine, FRANCE
Blumenfeld, Marta, Paris, FRANCE
Chumakov, Ilya, Vaux-le-Penil, FRANCE
Bougueleret, Lydie, Vanves, FRANCE
Bihain, Bernard, Encinitas, CA, United States
Essioux, Laurent, Paris, FRANCE
PATENT ASSIGNEE(S): Genset, FRANCE (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6476208	B1	20021105
APPLICATION INFO.:	US 2000-539333		20000330 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1999-416384, filed on 12 Oct 1999		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-126903P	19990330 (60)

	US 1999-131971P	19990430 (60)
	US 1999-132065P	19990430 (60)
	US 1999-143928P	19990714 (60)
	US 1999-145915P	19990727 (60)
	US 1999-146453P	19990729 (60)
	US 1999-146452P	19990729 (60)
	US 1999-162288P	19991028 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Fredman, Jeffrey
LEGAL REPRESENTATIVE: Saliwanchik, Lloyd & Saliwanchik
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Figure(s); 22 Drawing Page(s)
LINE COUNT: 10859
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 6 OF 7 USPATFULL on STN
TI Method for diagnosing a vascular condition
AB A method for diagnosing hypoxia, endothelial dysfunction, a vascular or circulatory condition of a subject, in which the level of expression of a gene, and/or the level of a metabolite or metabolic by-product in a biological test sample is measured and compared to a control sample so as to assess the vascular condition of the subject, is described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:198534 USPATFULL
TITLE: Method for diagnosing a vascular condition
INVENTOR(S): Adams, Michael A., Kingston, CANADA
Heaton, Jeremy P.W., Gananoque, CANADA
Graham, Charles H., Kingston, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002106634	A1	20020808
APPLICATION INFO.:	US 2002-59920	A1	20020129 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 1999-302554, filed on 30 Apr 1999, PATENTED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-83763P	19980501 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Licata & Tyrrell P.C., 66 East Main Street, Marlton, NJ, 08053
NUMBER OF CLAIMS: 50
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 26 Drawing Page(s)
LINE COUNT: 2176
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 7 OF 7 USPATFULL on STN
TI Method for diagnosing a vascular condition
AB A method for diagnosing hypoxia, endothelial dysfunction, a vascular or circulatory condition of a subject, in which the level of expression of a gene, and/or the level of a metabolite or metabolic by-product in a biological test sample is measured and compared to a control sample so as to assess the vascular condition of the subject, is described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:88195 USPATFULL
TITLE: Method for diagnosing a vascular condition
INVENTOR(S): Adams, Michael A., Kingston, CANADA

Heaton, Jeremy P. W., Gananoque, CANADA
Graham, Charles H., Kingston, CANADA
Brien, Susan E., Kingston, CANADA
PATENT ASSIGNEE(S): Queens University at Kingston, Kingston, CANADA
(non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6376169	B1	20020423
APPLICATION INFO.:	US 1999-302554		19990430 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-83763P	19980501 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Caputa, Anthony C.
ASSISTANT EXAMINER: Nickol, Gary B
LEGAL REPRESENTATIVE: Licata & Tyrrell P.C.
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 38 Drawing Figure(s); 26 Drawing Page(s)
LINE COUNT: 2141
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Refine Search

Search Results -

Terms	Documents
L6 and protein marker	89057

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L7

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DB=USPT; PLUR=YES; OP=OR			
L7	L6 and protein marker	89057	L7
L6	L5 and hypertension	3619	L6
L5	haptoglobin-1 precursor	117885	L5
L4	H factor 1	3583695	L4
L3	SNC73	0	L3
L2	L1 and hypertension	0	L2
L1	HAP-1	13	L1

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Search Results - Record(s) 1 through 10 of 3619 returned.

☐ 1. Document ID: US 6797708 B2

L6: Entry 1 of 3619 File: USPT Sep 28, 2004

US-PAT-NO: 6797708
DOCUMENT-IDENTIFIER: US 6797708 B2

TITLE: Inhibitors of cytosolic phospholipase A2

DATE-ISSUED: September 28, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McKew; John C.	Arlington	MA		
Tam; Steven Y.	Wellesley	MA		
Lee; Katherine L.	West Newton	MA		
Chen; Lihren	Cambridge	MA		
Thakker; Paresh	Boston	MA		
Sum; Fuk-Wah	Pomona	NY		
Behnke; Mark	Sommerville	MA		
Hu; Baihua	Audubon	PA		
Clark; James D.	Acton	MA		

US-CL-CURRENT: 514/228.2; 514/233.5, 514/254.09, 514/256, 514/278, 514/326, 514/327, 514/339, 514/345, 514/350, 514/357 , 514/364, 514/365, 514/374, 514/375, 514/381, 514/386, 514/406, 514/414, 544/143, 544/333, 544/373, 544/58.2, 546/115, 546/16, 546/201, 546/277.4, 548/125, 548/126, 548/181, 548/235, 548/236, 548/254, 548/311.4, 548/364.7, 548/407, 548/454, 548/455, 548/504

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 2. Document ID: US 6797695 B1

L6: Entry 2 of 3619 File: USPT Sep 28, 2004

US-PAT-NO: 6797695
DOCUMENT-IDENTIFIER: US 6797695 B1

TITLE: Human FGF-20 gene and gene expression products

DATE-ISSUED: September 28, 2004

h e b b g e e e f e ef b e

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Itoh; Nobuyuki	Kyoto			JP
Kavanaugh; Michael	Mill Valley	CA		

US-CL-CURRENT: 514/12; 530/350

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

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☐ 3. Document ID: US 6797502 B2

L6: Entry 3 of 3619 File: USPT Sep 28, 2004

US-PAT-NO: 6797502
DOCUMENT-IDENTIFIER: US 6797502 B2

TITLE: 18891, a novel human lipase

DATE-ISSUED: September 28, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kapeller-Libermann; Rosana	Chestnut Hill	MA		

US-CL-CURRENT: 435/198; 435/183, 435/195, 435/252.3, 435/320.1, 435/325, 530/350

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

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☐ 4. Document ID: US 6797499 B2

L6: Entry 4 of 3619 File: USPT Sep 28, 2004

US-PAT-NO: 6797499
DOCUMENT-IDENTIFIER: US 6797499 B2

TITLE: Isolated human dehydrogenases, nucleic acid molecules encoding these human dehydrogenases, and uses thereof

DATE-ISSUED: September 28, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Gong; Fangcheng	Germantown	MD		
Yan; Chunhua	Boyds	MD		
Di Francesco; Valentina	Rockville	MD		
Beasley; Ellen M.	Darnestown	MD		

US-CL-CURRENT: 435/189

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Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 5. Document ID: US 6797494 B1

L6: Entry 5 of 3619 File: USPT Sep 28, 2004

US-PAT-NO: 6797494
DOCUMENT-IDENTIFIER: US 6797494 B1

TITLE: Self-replicating episomal expression vectors conferring tissue-specific gene expression

DATE-ISSUED: September 28, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Antoniou; Michael	Edgeware			GB
Grosveld; Frankin G.	Rotterdam			NL

US-CL-CURRENT: 435/70.1; 435/320.1, 435/455

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 6. Document ID: US 6794363 B2

L6: Entry 6 of 3619 File: USPT Sep 21, 2004

US-PAT-NO: 6794363
DOCUMENT-IDENTIFIER: US 6794363 B2

TITLE: Isolated amyloid inhibitor protein (APIP) and compositions thereof

DATE-ISSUED: September 21, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bejanin; Stephane	Paris			FR
Tanaka; Hiroaki	Antony			FR

US-CL-CURRENT: 514/12; 435/23, 530/350, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 7. Document ID: US 6794362 B1

L6: Entry 7 of 3619 File: USPT Sep 21, 2004

US-PAT-NO: 6794362
DOCUMENT-IDENTIFIER: US 6794362 B1

h e b b g e e e f e ef b e

TITLE: Asparagine containing elastin peptide analogs

DATE-ISSUED: September 21, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Sandberg; Lawrence B.	Colton	CA		
Mitts; Thomas F.	Visalia	CA		
Jimenez, Jr.; Felipe	Loma Linda	CA		

US-CL-CURRENT: 514/11; 424/404, 424/455, 424/489, 514/16, 514/17, 514/18, 530/317, 530/328, 530/329, 530/330

☐ 8. Document ID: US 6794160 B1

L6: Entry 8 of 3619 File: USPT Sep 21, 2004

US-PAT-NO: 6794160
DOCUMENT-IDENTIFIER: US 6794160 B1

TITLE: Hormone receptor compositions and methods

DATE-ISSUED: September 21, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Evans; Ronald M.	La Jolla	CA	92037	
Weinberger; Cary A.	San Diego	CA	92129	
Hollenberg; Stanley M.	Seattle	WA	98103	
Giguere; Vincent	Etobicoke, Ont.		M9A 5C6	CA
Arriza; Jeffrey	Durham	NC	27704	
Thompson; Catherine C.	Malverne	NY	11565	
Ong; Estelita S.	San Diego	CA	92117	

US-CL-CURRENT: 435/69.1; 435/252.3, 435/320.1, 536/23.5

☐ 9. Document ID: US 6794143 B2

L6: Entry 9 of 3619 File: USPT Sep 21, 2004

US-PAT-NO: 6794143
DOCUMENT-IDENTIFIER: US 6794143 B2

TITLE: Biallelic markers derived from genomic regions carrying genes involved in arachidonic acid metabolism

h e b b g e e e f e ef b e

DATE-ISSUED: September 21, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Blumenfeld; Marta	Paris			FR
Bougueleret; Lydie	Vanves			FR
Chumakov; Ilya	Vaux-le-Penil			FR
Cohen; Annick	Paris			FR

US-CL-CURRENT: 435/6; 435/91.2, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 10. Document ID: US 6790965 B1

L6: Entry 10 of 3619 File: USPT Sep 14, 2004

US-PAT-NO: 6790965
DOCUMENT-IDENTIFIER: US 6790965 B1

TITLE: Combinatorial dihydrobenzopyran library

DATE-ISSUED: September 14, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Baldwin; John J.	Gwynedd Valley	PA		
Reader; John C.	Princeton	NJ		
Dillard; Lawrence W.	Hopewell	NJ		
Li; Ge	Plainsboro	NJ		
Zeng; Wenguang	Lawrenceville	NJ		

US-CL-CURRENT: 549/32; 436/518, 436/524, 436/525, 436/526, 436/527, 436/528, 436/529, 436/530, 436/531, 549/265, 549/40 , 549/404, 549/408, 564/183, 564/184, 564/186

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

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Terms	Documents
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